INDEXED TO ALL 4 (NICOTINE, CAFFEINE, THEOPHYLLINE, & THEOBROMINE)

FROM EMBASE {*Excerpta Medica*; DIALOG: File 73}

1

AN- <DIALOG> 2331531
TI- Interactions between caffeine, other drinks, tobacco and drugs
TI- <ORIGINAL> WECHSELWIRKUNGEN ZWISCHEN COFFEIN, ANDEREN GENUSSMITTELN UND ARZNEISTOFFEN
AU- Czok G.
CS- Pharmakol. Inst., Univ., 2000 Hamburg 20
CS- GERMANY, WEST
SO- <JN> Z. ERNAHRUNGSWISS.
CP- GERMANY, WEST
SO- <PY> 1980
SO- <VO> 19/4 (290-295)
LA- GERMAN
SL- ENGLISH
AB- Caffeine is ingested not only with beverages as coffee, tea, coca-cola but also in form of many analgetic drugs. Therefore interactions of this substance with other biologically active substances and drugs should be expected, and the knowledge of these would be of practical importance. The interactions between caffeine and alcohol, smoking, salicylic acid, phenacetin, barbiturates, and theobromine are described.

2

AN- <DIALOG> 2062364
TI- Biological psychiatric aspects of dependence on tobacco and coffee
AU- Hoes M.J.A.J.M.
CS- Bethesdaziekenh., 4000 AA Tiel
CS- NETHERLANDS
SO- <JN> TIJDSCHR. ALCOHOL DRUGS ANDERE PSYCHOTROPE STOFF.
CP- NETHERLANDS
SO- <PY> 1980
SO- <VO> 6/2 (48-51)
LA- DUTCH
SL- ENGLISH
AB- Tobacco and coffee are, with alcohol, the most widely used drugs of pleasure. The use of tobacco and coffee can deteriorate to dependence also. The withdrawal effects from tobacco and coffee are however of little clinical significance, the subjective effects during withdrawal being the most impressive. The pharmacologically most important active principle is in tobacco the nicotine, in coffee the caffeine. Because of their physiological, psycho-, and neuropharmacological effects, nicotine and caffeine can be classified as stimulants. Stimulants are potent pharmacological reinforcers. By virtue of this property of nicotine and caffeine one can explain the occurrence of dependence on tobacco and coffee use.

2050798016

Source: https://www.industrydocuments.ucsf.edu/docs/hlgl0000

3

AN- <DIALOG> 1141010
TI- Role of tobacco smoking in pharmacokinetics
AU- Jusko W.J.
CS- Dept. Pharmaceut., Sch. Pharm., State Univ. New York, Buffalo, Amherst, N.Y. 14260
CS- USA
SO- <JN> J. PHARMACOKINET. BIOPHARM.
CP- USA
SO- <PY> 1978
SO- <VO> 6/1 (7-39)
LA- ENGLISH
AB- The pervasiveness of tobacco use in our society and the frequency of altered disposition and pharmacological effects of many common therapeutic and recreational drugs in smokers make it apparent that the smoking habit should be considered as one of the primary sources of drug interactions in man. Most of the experimental work in man, animals, and tissue on enzyme systems indicates that the dominant effect of smoking is enhanced disposition caused by induction of hepatic microsomal enzymes. The primary causal agents are probably the polynuclear aromatic hydrocarbons, which are potent and persistent in tissues. While several of the hepatic microsomal drug-metabolizing enzymes are stimulated in smokers, the selectivity of this enhancement in activity is unpredictable and the effects of tobacco smoke on other potential rate-limiting disposition processes are largely unexplored.

2050798017

Source: https://www.industrydocuments.ucsf.edu/docs/hlgl0000

CAFFEINE & NICOTINE AND EEG, ERP, EP, ETC.

FROM psycINFO {DIALOG: FILE 11}

4

AN- 00167422

TI- The effect of caffeine, nitrazepam and cigarette smoking on the contingent negative variation in man.

AU- Ashton, Heather Millman, J. E. Telford, Rosemary Thompson, J. W.

CS- U. Newcastle upon Tyne, Clinical Pharmacology Unit, England

SO- <JN> Electroencephalography & Clinical Neurophysiology

SO- 1974 Jul Vol. 37(1) 59-71

LA- English

DT- JOURNAL ARTICLE

AB- Studied the effects of caffeine, nitrazepam, and cigarette smoking on the contingent negative variation (CNV) in college students and staff. In 13 Ss who took 300 mg of caffeine citrate, the mean magnitude of CNV was significantly increased 35-42 min later; in 12 Ss who took 2.5 mg of nitrazepam, mean magnitude of the CNV was significantly decreased after 35-42 min. In 22 smokers, significant changes in CNV magnitude were also observed after smoking 1 cigarette. However, the direction of change in CNV size following smoking differed in different smokers beyond the chance level; some smokers showed consistent increases and others showed consistent decreases in CNV magnitude. These changes were interpreted as reflecting stimulant and depressant effects. Correlations of the percentage change in CNV magnitude with rate of nicotine intake and with extraversion (measured by the Eysenck Personality Inventory) suggests that the rate of nicotine intake in extraverted smokers was slower and associated with a stimulant effect, while in introverted smokers the rate was faster and associated with a depressant effect. There was a significant negative correlation in smokers between reaction time and CNV magnitude. There was no significant overall correlation between visual evoked response amplitude and CNV magnitude. (French summary) (46 ref)

AB- (PsycINFO Database Copyright 1974 American Psychological Assn, all rights reserved)

Source: https://www.industrydocuments.ucsf.edu/docs/hlgl0000

FROM EMBASE {*Excerpta Medica*; DIALOG: File 73}

5

AN- <DIALOG> 9177011
TI- Caffeine antagonizes EEG effects of tobacco withdrawal
AU- Cohen C. Pickworth W.B. Bunker E.B. Henningfield J.E.
CS- Clinical Pharmacology Laboratory, National Inst. on Drug Abuse, Addiction Research Center, 4940 Eastern Avenue, Baltimore, MD 21224
CS- USA
SO- <JN> PHARMACOL. BIOCHEM. BEHAV.
CP- USA
SO- <PY> 1994
SO- <VO> 47/4 (919-926)
SN- 0091-3057
LA- English
SL- English
AB- Six current cigarette smokers and coffee drinkers were given combinations of 0, 150, or 300 mg caffeine and 0, 2, or 4 mg nicotine polacrilex following 12-h nicotine and caffeine abstinence. On one study day, subjects were allowed to smoke cigarettes and to drink caffeinated beverages and no drugs were given. Tobacco and caffeine abstinence impaired performance on the serial addition/subtraction and digit recall tasks; decreased scores on the MBG scale and ratings of 'clear-headed' and 'quick-witted'; and increased ratings of 'irritability,' 'muscular tension,' 'headache,' 'drowsy,' 'clumsy,' 'feeble,' and 'dreamy.' The deprivation caused characteristic EEG signs of nicotine withdrawal including increased theta power and decreased alpha frequency. These EEG effects were reversed by cigarette smoking and caffeine administration, but nicotine polacrilex was less effective. Deprivation-induced performance and subjective measures were not changed by administration of nicotine and caffeine combinations.

Source: https://www.industrydocuments.ucsf.edu/docs/hlgl0000

6

AN- <DIALOG> 8818236
TI- Drug effects on the human EEG
AU- Holmes G.L. Korteling F.
CS- Clinical Neurophysiology Laboratory, Children's Hospital, Harvard Medical School, 300 longwood Avenue, Boston, MA 02115
CS- USA
SO- <JN> AM. J. EEG TECHNOL.
CP- USA
SO- <PY> 1993
SO- <VO> 33/1 (1-26)
SN- 0002-9238
LA- English
SL- English
AB- The effects of drugs on the EEG have been of interest since Berger demonstrated in 1929 that electrical signals could be recorded from the scalp. Berger described changes in the EEG following a variety of medications and, based on these observations, hypothesized that there was a relationship between changes in alpha activity and mental processes. A large number of drugs have now been demonstrated to alter the EEG and many of these drug-induced changes may be clinically useful. Understanding the effects and significance of drug effects on the EEG requires an understanding of the physiological basis of electroencephalography. This paper reviews the physiological basis of the EEG and the effects of commonly used drugs on background rhythms and epileptiform activity. The effects of prenatal and postnatal drug exposure on the neonatal EEG are also reviewed.

2050798020

Source: https://www.industrydocuments.ucsf.edu/docs/hlgl0000

7

AN- <DIALOG> 5334019
TI- Chemistry and pharmacology of nootropics
AU- Nicolaus B.J.R.
CS- ISF Inst. Biomed. Res., 20090 Trezzano S/N (Milano)
CS- ITALY
SO- <JN> DRUG DEV. RES.
CP- USA
SO- <PY> 1982
SO- <VO> 2/5 (463-474)
LA- ENGLISH
AB- The paper discusses the chemistry and pharmacology of the nootropics, reviewing the new chemical entities to recently emerge and in particular the five-membered heterocyclic lactams belonging to the pyrrolidinone class, such as piracetam and oxiracetam. These gamma lactams are related to the folded conformation of GABA, which seems to play a role in the uptake mechanisms - that is, in the transport of GABA across the membranes into different nervous tissue elements. The classification of nootropics is discussed, taking into consideration the six main criteria recently suggested, which are: no direct vasoactivity, no change in basic rhythm of EEG activity, blood brain barrier passage, positive metabolic activity in humans and animals, low incidence of side-effects, and objective demonstration of clinivicacy. A critical analysis is made of the various pharmacological and biochemical models employed to screen the nootropics, including a new approach based on the impaired learning rate of spontaneously hypertensive rats with cerebrovascular lesions. The activity of the new GABA-derivative nootropic called oxiracetam is also described and compared with the forerunner of the class, piracetam.
ID- 43 literature references

8

AN- <DIALOG> 5321840
TI- Contingent negative variation (CNV) and drug effects
AU- Ashton H. Marsh V.R. Thompson J.W.
CS- Clin. Psychopharmacol. Unit, Univ. Newcastle upon Tyne, Newcastle upon Tyne NE1 7RU
CS- UNITED KINGDOM
SO- <JN> BIOL. PSYCHOL.
CP- NETHERLANDS
SO- <PY> 1982
SO- <VO> 15/3-4 (293-294)
LA- ENGLISH

2050298021

Source: https://www.industrydocuments.ucsf.edu/docs/hlgl0000

9

AN- <DIALOG> 2303901
TI- The EEG effects of tobacco smoking - a review
AU- Conrin J.
CS- W.A. Howe Developm. Cent., Tinley Park, Ill. 60477
CS- USA
SO- <JN> CLIN. EEG
CP- USA
SO- <PY> 1980
SO- <VO> 11/4 (180-187)
LA- ENGLISH
ID- review short survey

10

AN- <DIALOG> 970521
TI- The use of event related slow potentials of the human brain as an objective method to study the effects of centrally acting drugs
AU- Ashton H. Millman J.E. Telford R. Thompson J.W.
CS- Clin. Psychopharmacol. Unit, Dept. Pharmacol. Scis, Univ. Newcastle upon Tyne
CS- UNITED KINGDOM
SO- <JN> NEUROPHARMACOLOGY
CP- ENGLAND
SO- <PY> 1977
SO- <VO> 16/7-8 (531-532)
LA- ENGLISH

11

AN- <DIALOG> 852122
TI- Interactions of Deltasup 9 tetrahydrocannabinol with other pharmacological agents
AU- Consroe P. Jones B. Laird H.
CS- Dept. Pharmacol. Toxicol., Univ. Arizona Coll. Pharm., Tucson, Ariz. 85721
CS- USA
SO- <JN> ANN.N.Y.ACAD.SCI.
CP- USA
SO- <PY> 1976
SO- <VO> Vol.281 (198-211)
LA- ENGLISH
AB- The present paper provides data on the interactive effects of THC with a number of other drugs in two agonist antagonist paradigms in the rabbit.

2050798022

Source: https://www.industrydocuments.ucsf.edu/docs/hlgl0000

12

AN- <DIALOG> 399503
TI- Central effects of a series of Nsup 1, Nsup 4 acyl piperazine derivatives
AU- Georgiev V. Petkov V. Petkova B.
CS- Dept. Pharmacol., Inst. Physiol., Bulg. Acad. Scis, Sofia
CS- BULGARIA
SO- <JN> ACTA PHYSIOL.PHARMACOL.BULG.
CP- BULGARIA
SO- <PY> 1974
SO- <VO> Vol. 1 (54-64)
LA- ENGLISH

13

AN- <DIALOG> 256590
TI- Stimulant and depressant effects of cigarette smoking on brain activity in man
AU- Ashton H. Millman J.E. Telford R. Thompson J.W.
CS- Clin. Pharmacol. Unit, Dept. Pharmacol., Univ. Newcastle upon Tyne
CS- UNITED KINGDOM
SO- <JN> BRIT.J.PHARMACOL.
CP- ENGLAND
SO- <PY> 1973
SO- <VO> 48/4 (715-717)
LA- ENGLISH
AB- Cigarette smoking was found to alter the E.E.G. negativity associated with attention: the 'contingent negative variation' was increased in some smokers and decreased in others. This finding is consistent with the known dual action of nicotine to stimulate or depress neural activity and suggests a means of investigating the effects of other centrally acting drugs on the human brain.

2050798023

Source: https://www.industrydocuments.ucsf.edu/docs/hlgl0000

FROM MEDLINE {DIALOG: FILE 155}

14

AN- <DIALOG> 08782702
TI- Cholinergic responses in cloned human TE671/RD tumour cells.
AU- Grassi F Giovannelli A Fucile S Mattei E Eusebi F
CS- Dipartimento di Medicina Sperimentale, Universita di Roma La Sapienza, Italy.
SO- <JN> Pflugers Arch
CP- GERMANY
SO- <PY> Oct 1993
SO- <VO> 425 (1-2) p117-25
SN- 0031-6768
LA- ENGLISH
DT- JOURNAL ARTICLE
AB- The cholinergic responses of the human tumour cell line TE671/RD were examined using digital Ca2+ imaging fluorescence microscopy and patch-clamp measurements. In response to stimulation of the muscarinic acetylcholine (ACh) receptor (mAChR), the intracellular concentration of Ca2+ ([Ca2+]i) rose about two-fold, in parallel with inositol 1,4,5-trisphosphate accumulation, measured by chromatographic techniques. By contrast, there was no increment of [Ca2+]i upon stimulation of the nicotinic ACh receptor (nAChR), nor after caffeine application. Electrophysiological experiments showed that TE671/RD cells lack functional voltage-activated Ca2+ channels. The stimulation of the nAChR induced transient whole-cell currents (IACh). Little or no current was detected in isotonic extracellular Ca2+, with Cs+ in the patch pipette. Cell pretreatment with muscarine reduced IACh by about 20%, without consistent modifications of current kinetics. Muscarine applied to the extra-patch membrane under the cell-attached configuration had no obvious effect on ACh-evoked unitary events. In conclusion, in human TE671/RD cells, muscarinic stimulation increases [Ca2+]i, while nicotinic stimulation does not. In addition, the nAChR exhibits peculiar ion permeability properties and is not functionally regulated by the breakdown of phosphoinositides.

15

AN- <DIALOG> 02621222
TI- Pharmacological evaluation of a new Ca++ antagonist, 8-(N,N-diethylamino)octyl 3,4,5-trimethoxybenzoate hydrochloride (TMB-8): studies in skeletal muscles.
AU- Malagodi MH Chiou CY
SO- <JN> Pharmacology
CP- SWITZERLAND
SO- <PY> 1974
SO- <VO> 12 (1) p20-31
SN- 0031-7012
LA- ENGLISH
DT- JOURNAL ARTICLE

2050798024

Source: https://www.industrydocuments.ucsf.edu/docs/hlgl0000

16

AN- <DIALOG> 02270227
TI- Effects of nicotine and arousal on the monkey electroencephalogram.
AU- Cheshire PJ Kellett DN Willey GL
SO- <JN> Experientia
CP- SWITZERLAND
SO- <PY> May 1 1973
SO- <VO> 29 (1) p71-3
SN- 0014-4754
LA- ENGLISH
DT- JOURNAL ARTICLE

FROM BIOSIS PREVIEWS {DIALOG: FILE 5}

17

AN- 1629474
TI- MODERN PROBLEMS OF PHARMACO PSYCHIATRY VOL 8 PSYCHOTROPIC DRUGS AND THE HUMAN ELECTRO ENCEPHALOGRAM
AU- ITIL T M
SO- <JN> MOD PROBL PHARMACOPSYCHIATR
SO- <PY> 1974 377.
SO- <JN> Modern Problems of Pharmacopsychiatry
DT- BOOK

Source: https://www.industrydocuments.ucsf.edu/docs/hlgl0000

FROM SCISEARCH {ISI's *Science Citation Index*; DIALOG: File 434}

18

GA- NV149
NR- 14
TI- PRETASK SMOKING AND SMOKING DURING A TASK - EFFECTS ON COGNITIVE PERFORMANCE, PUFFING BEHAVIOR, PHYSIOLOGICAL AND SUBJECTIVE PARAMETERS
AU- HASENFRATZ M BATTIG K
CS- ETH ZURICH,INST TOXICOL,DEPT BEHAV BIOL,SCHORENSTR 16/CH-8603 SCHWERZENBACH//SWITZERLAND/
SO- <JN> HUMAN PSYCHOPHARMACOLOGY-CLINICAL AND EXPERIMENTAL
SO- <PY> 1994
SO- <VO> 9
SO- <IS> N3
SO- <IS> MAY-JUN
SO- <PG> 181-189
SN- 0885-6222
LA- ENGLISH
DT- ARTICLE
AB- Twenty female regular smokers participated in four experimental sessions, each of which involved performing a rapid information processing task (RIP) twice. According to a 2 x 2 cross-over design the subjects were allowed to either real or sham smoke a single habitual cigarette between the two RIP trials and to real or sham smoke ad libitum during the second RIP trial. RIP performance was significantly better when the smokers were allowed to smoke during the task. On the other hand, the improvement after pretask smoking failed to reach significance. The puffing intervals were similar for real and sham smoking during rest but significantly longer with real than with sham smoking during the task. The increases in electrocortical and cardiovascular arousal as well as the subjective effects were similar for pretask and task smoking. After pretask smoking these effects vanished for the subsequent task period, whereas they were maintained but not further increased when pretask smoking was followed by task smoking. It was concluded that mental performance was increased but overarousal was avoided by accurate nicotine titration during the RIP task.

2050798026

Source: https://www.industrydocuments.ucsf.edu/docs/hlgl0000

19

GA- KV797
NR- 116
TI- STIMULANT-DRUGS AND VIGILANCE PERFORMANCE - A REVIEW
AU- KOELEGA HS
CS- UNIV UTRECHT,DEPT PSYCHOPHARMACOL,HEIDELBERGLAAN 2/3584 CS UTRECHT//NETHERLANDS/ UNIV UTRECHT,PSYCHOL LAB/3584 CS UTRECHT//NETHERLANDS/
SO- <JN> PSYCHOPHARMACOLOGY
SO- <PY> 1993
SO- <VO> 111
SO- <IS> N1
SO- <IS> APR
SO- <PG> 1-16
SN- 0033-3158
LA- ENGLISH
DT- REVIEW
AB- The literature on the effects of some stimulant drugs (amphetamine, methylphenidate, caffeine, and nicotine) on vigilance performance is reviewed. Improvement of overall level of performance (both accuracy and speed) after the intake of amphetamine, caffeine, and nicotine has often been reported, and the decrement in performance with time has been shown to be prevented especially with amphetamine and nicotine. Effects on false alarms are negligible. In studies where a test battery was employed, vigilance tasks appeared to be the most sensitive performance tests in detecting the effects of stimulants; however, different vigilance tasks may measure different aspects of mental functions. There is no support for earlier conclusions that improvements are noticed only in fatigued subjects in protracted sessions. Evidence from several studies does not support the hypothesis that improvements are only a recovery of withdrawal-induced impairment. Because positive effects have been obtained with drugs possessing different mechanisms of action, there is as yet no clear support for a noradrenergic, dopaminergic, or cholinergic theory of sustained attention. Simple neurotransmitter theories of attention and information processing may be untenable.

20

GA- DR204
NR- 562
TI- THE PHARMACOLOGY OF HUMAN ANXIETY
AU- NUTT DJ
CS- SCH MED SCI BRISTOL,RECKITT & COLMAN PSYCHOPHARMACOL UNIT,UNIV WALK/BRISTOL BS8 1TD//ENGLAND/
SO- <JN> PHARMACOLOGY & THERAPEUTICS
SO- <PY> 1990
SO- <VO> 47
SO- <IS> N2
SO- <PG> 233-266
LA- ENGLISH
DT- REVIEW

2050798027

Source: https://www.industrydocuments.ucsf.edu/docs/hlgl0000

FROM CA SEARCH {Abstractless *Chemical Abstracts*; DIALOG: File 399}

21

AZ- 82038293
DT- JOURNAL
TI- Electroencephalographic effects of caffeine, nicotine, tobacco smoking, and alcohol
AU- Murphree, Henry B.
CS- Rutgers Med. Sch. Coll. Med. Dent. New Jersey Piscataway N. J.
JN- Mod. Probl. Pharmacopsychiatry
PY- 1974
VO- 8,
PG- 22-36
LA- English

2050798028

Source: https://www.industrydocuments.ucsf.edu/docs/hlgl0000

NICOTINE & EEG/EP/ERP ETC. IN HEALTHY/NORMAL VOLUNTEERS/SUBJECTS

FROM psycINFO {DIALOG: FILE 11}

22

AN- 00860451
TI- Nicotine as a cognitive enhancer.
AU- Warburton, David M.
CS- U Reading, England
SO- <JN> Progress in Neuro-Psychopharmacology & Biological Psychiatry
SO- 1992 Mar Vol 16(2) 181-191
SN- 02785846
LA- English
DT- JOURNAL ARTICLE
AB- A review of the literature shows that in healthy volunteers, nicotine improves sustained attention, attentional processing, and selective attention. Another indicator of improved information processing with nicotine is the changes observed in event-related potentials. Nicotine also improves immediate and long-term memory in healthy volunteers. Patients with senile dementia of the Alzheimer type have demonstrated improved information processing with nicotine.
AB- (PsycINFO Database Copyright 1992 American Psychological Assn, all rights reserved)

2050798029

Source: https://www.industrydocuments.ucsf.edu/docs/hlgl0000

FROM EMBASE {*Excerpta Medica*; DIALOG: File 73}

23

AN- <DIALOG> 9052631
TI- Normalization of auditory physiology by cigarette smoking in schizophrenic patients
AU- Adler L.E. Hoffer L.D. Wiser A. Freedman R.
CS- Department of Psychiatry, Colorado Univ. Health Sciences Ctr., Box C268-16, 4200 East Ninth Ave., Denver, CO 80262
CS- USA
SO- <JN> AM. J. PSYCHIATRY
CP- USA
SO- <PY> 1993
SO- <VO> 150/12 (1856-1861)
SN- 0002-953X
LA- English
SL- English
AB- Objective: Because many schizophrenic patients are heavy smokers, it has been suggested that nicotine normalizes some neuronal deficit involved in their illness. Schizophrenic subjects have various difficulties with maintenance of attention and selective processing of sensory information. One defect in sensory gating in schizophrenia has been characterized by recording auditory evoked potentials. Most normal subjects have a decrease in the evoked response to the second of two closely paired stimuli, whereas most schizophrenic subjects do not. The aim of this study was to determine whether smoking normalizes this deficit in auditory sensory gating in schizophrenia. Method: Changes in auditory sensory gating in response to smoking cigarettes were studied in 10 smokers without psychiatric illness and 10 schizophrenic smokers. Both groups were asked to abstain from smoking from 11:00 p.m. until 8:00 a.m. the next morning, when their auditory evoked responses to pairs of clicks were recorded. The ability to gate sensory information is reflected in a decrease in the P50 wave amplitude in response to the second of the two stimuli. After baseline recordings, the subjects smoked as much as they wished, and then two postsmoking recordings were performed. Results: The schizophrenic patients had a marked but brief improvement in P50 auditory gating immediately after smoking, whereas P50 gating for the normal smokers was slightly impaired. Conclusions: This study suggests that cigarette smoking can transiently normalize the impairment of auditory sensory gating in schizophrenic patients.

2050798030

Source: https://www.industrydocuments.ucsf.edu/docs/hlgl0000

24

AN- <DIALOG> 8249098
TI- Effects of cigarette smoking upon frequencies of EEG alpha rhythm and finger tapping
AU- Roth N. Battig K.
CS- Laboratory of Comparative Physiology and Behavioral Biology, Swiss Federal Institute of Technology, ETH-Zentrum, Turnerstrasse 1, CH-8092 Zurich
CS- Switzerland
SO- <JN> PSYCHOPHARMACOLOGY
CP- Germany
SO- <PY> 1991
SO- <VO> 105/2 (186-190)
SN- 0033-3158
ON- 003331589100264C
LA- English
AB- Under the influence of nicotine, an increase in EEG alpha frequency as well as finger tapping speed has been reported from separate experiments, and it has also been shown that tapping and alpha frequencies may correlate in defined experimental settings. The present study aimed at the analysis of smoking effects using parallel recordings of both EEG and finger tapping. Twelve healthy subjects were tested in two sessions (smoking deprivation versus smoking). After smoking, both dominant alpha frequency ($P < 0.05$) and tapping performance ($P < 0.001$) were higher than during deprivation. In most subjects, the increase of both parameters developed in parallel, and during phases of very stable tapping the ratio of alpha and tapping frequencies was close to 2:1. Both frequencies correlated during the deprived ($r = 0.6108$, $P = 0.035$) as well as the smoking ($r = 0.7009$, $P = 0.011$) conditions. Results confirm earlier findings regarding the effect of smoking upon EEG and tapping, and the parallel changes of both parameters may be attributed to the pharmacological properties of nicotine. Besides possible peripheral and spinal effects of nicotine, the increase of tapping performance parallels the increased frequency of central nervous pacemakers.

2050798031

Source: https://www.industrydocuments.ucsf.edu/docs/hlgl0000

25

AN- <DIALOG> 5291269
TI- Effects of nicotine on the visual evoked response
AU- Woodson P.P. Baettig K. Etkin M.W. et al.
CS- Swiss Fed. Inst. Technol., Zurich
CS- SWITZERLAND
SO- <JN> PHARMACOL. BIOCHEM. BEHAV.
CP- USA
SO- <PY> 1982
SO- <VO> 17/5 (915-920)
LA- ENGLISH
AB- The effects of smoking cigarettes differing in nicotine content (0.14 vs 1.34 mg/cigarette) on the peak-to-peak amplitude and peak latency of the human averaged visual evoked response (AVER) were measured in 10 male smokers after a 2-hr smoking deprivation period. The AVER was obtained under five different flash intensities. Eight different peaks were involved in the amplitude and latency measurements. The nicotine dosage and flash intensity factors both had significant effects on peak-to-peak amplitudes while only the flash intensity factor affected peak latencies. The general enhancement of peak-to-peak amplitudes by the 1.34 mg cigarette, relative to the 0.14 mg cigarette, indicates that the effects of cigarette smoking on the AVER are predominantly due to nicotine's psychopharmacologic action, as opposed to other elements in tobacco smoke or as opposed to nonpharmacologic mechanisms involving learning processes. Past research, on an electrophysiological and behavioral level, indicating that nicotine, as administered via cigarette smoking, may have enhancing and/or restorative effects on visual attentional processes in the quiescent smoker was supported.

2050798032

Source: https://www.industrydocuments.ucsf.edu/docs/hlgl0000

26

AN- <DIALOG> 2134192
TI- Biphasic dose-related responses of the CNV (contingent negative variation) to I.V. nicotine in man
AU- Ashton H. Marsh V.R. Millman J.E. et al.
CS- Clin. Psychopharmacol. Unit, Dept. Pharmacol. Sci. Univ., Newcastle-upon Tyne NE24AB
CS- UNITED KINGDOM
SO- <JN> BR. J. CLIN. PHARMACOL.
CP- ENGLAND
SO- <PY> 1980
SO- <VO> 10/6 (579-589)
LA- ENGLISH
AB- The effects of intravenous injections of nicotine bitartrate, given as intermittent 'shots', on the magnitude of the contingent negative variation (CNV) were studied in twelve male volunteers. In one series of experiments in five subjects, a fixed dose of nicotine was used. In three of these subjects nicotine 500 mug or 750 mug produced a reproducible increase in magnitude of the CNV. In two subjects nicotine 750 mug produced a reproducible decrease in magnitude of the CNV. The direction and magnitude of the CNV changes could be reproduced by cigarette smoking. In another series of experiments in eight subjects, dose-response relationships for the effect of nicotine on the CNV were measured using a range of doses from 12.5 to 800 mug. Individual and mean dose-response curves were found to be biphasic so that whilst smaller doses produced an increase of CNV magnitude (stimulant effect), larger doses produced a decrease of the CNV (depressant effect). The results are discussed in relation to the possible mechanism of action on the brain of nicotine as obtained by inhaling cigarette smokers.

2050798033

Source: https://www.industrydocuments.ucsf.edu/docs/hlgl0000

FROM MEDLINE {DIALOG: FILE 155}

27

AN- <DIALOG> 08762392
TI- The effect of nicotine on recurrent inhibition in the spinal cord.
AU- Shefner JM Berman SA Young RR
CS- Spinal Cord Injury Service, Brockton/West Roxbury VA Medical Center, MA.
SO- <JN> Neurology
CP- UNITED STATES
SO- <PY> Dec 1993
SO- <VO> 43 (12) p2647-51
SN- 0028-3878
LA- ENGLISH
DT- JOURNAL ARTICLE
AB- Recurrent inhibition via Renshaw cells provides a mechanism by which spinal and supraspinal centers exert control over movement. The conditioned H-reflex technique of Pierrot-Deseilligny and Bussel permits noninvasive assessment of recurrent inhibitory pathways. We employed this technique to investigate changes in Renshaw cell activity due to nicotine (a potent CNS cholinergic agonist that excites Renshaw cells in animals) contained in inhaled tobacco smoke. In 10 normal subjects, cigarette smoking caused a large, rapid drop in the conditioned H-response amplitude, implying increased activation of Renshaw cells. The time course of the change in conditioned H-response amplitude closely approximated the known pharmacokinetics of inhaled nicotine. Nicotine administered via chewing gum had a much slower and less dramatic effect, probably due to the slower rise in blood levels with this mode of administration. Increased activity in Renshaw cells may contribute to spasticity in spinal cord-injured patients, raising the possibility that cigarette smoking could cause further increases in tone in such patients.

FROM SCISEARCH {ISI's *Science Citation Index*; DIALOG: File 434}

28

GA- CR240
NR- 32
TI- EFFECTS OF NICOTINE GUM ON PSYCHOMOTOR PERFORMANCE IN SMOKERS AND NONSMOKERS
AU- HINDMARCH I KERR JS SHERWOOD N
CS- UNIV LEEDS,DEPT PSYCHOL,HUMAN PSYCHOPHARMACOL RES UNIT/LEEDS LS2 9JT/W YORKSHIRE/ENGLAND/
SO- <JN> PSYCHOPHARMACOLOGY
SO- <PY> 1990
SO- <VO> 100
SO- <IS> N4
SO- <PG> 535-541
LA- ENGLISH
DT- ARTICLE

2050298034

Source: https://www.industrydocuments.ucsf.edu/docs/hlgl0000

NICOTINE EP/EEG/ERP ETC. REVIEWS, BOOK CHAPTERS, MONOGRAPHS, ETC.

FROM EMBASE {*Excerpta Medica*; DIALOG: File 73}

29

AN- <DIALOG> 629172
TI- The effect of drugs on the electroencephalogram: a review with commentary
AU- McFarland H.R.
CS- Univ. Kansas Med. Cent., Kansas City, Kans.
CS- USA
SO- <JN> CLIN.EEG (CHICAGO)
CP- USA
SO- <PY> 1975
SO- <VO> 6/4 (178-183)
LA- ENGLISH
AB- An outline of the salient effects of various centrally active drugs on the EEG of humans is presented and discussed. The characteristic EEG patterns for phenothiazines, anticholinesterases, anticholinergics, thymoleptics, hallucinogenics, antihistamines, stimulants, narcotics, alcohol, hormones, nicotine and cannabis are described. Although there are difficulties in obtaining reliable, comparable, specific EEG modifications for different drugs, it is essential that electroencephalographers continue to seek this information. An elemental EEG drug protocol is included for use in future investigation.

Source: https://www.industrydocuments.ucsf.edu/docs/hlgl0000

FROM MEDLINE {DIALOG: FILE 155}

30

AN- <DIALOG> 08794554
TI- NGF and the treatment of Alzheimer's disease.
AU- Olson L
CS- Department of Neuroscience, Karolinska Institute, Stockholm, Sweden.
SO- <JN> Exp Neurol
CP- UNITED STATES
SO- <PY> Nov 1993
SO- <VO> 124 (1) p5-15
SN- 0014-4886
LA- ENGLISH
DT- JOURNAL ARTICLE REVIEW REVIEW, ACADEMIC
AB- Issues related to the possible treatment of Alzheimer's disease with nerve growth factor (NGF) are discussed. Animal research has demonstrated that the ascending cholinergic projections in the brain express low- and high-affinity receptors for NGF and are NGF-sensitive as well as probably NGF-dependent. Cholinergic lesions lead to cognitive disturbances, and treatment with NGF can improve cognitive behavior in animals. It thus seems reasonable to attempt to counteract the degeneration of cholinergic systems known to occur in patients with Alzheimer's disease by treatment with NGF. There are several different possible ways of stimulating NGF receptors such as NGF infusion, implantation of slow-release biodegradable pellets, using carrier-mediated transport across the blood-brain barrier, grafting NGF-producing cells, transferring genes directly to the brain, developing NGF receptor agonists, or controlling the endogenous NGF production. The first clinical trial of NGF infusion is described in some detail. Based on background information from intracerebral infusion of NGF in parkinsonian patients, attempting to support intraputaminal chromaffin tissue grafts, a study was initiated using a radio-controlled fully implantable pumping device delivering NGF to the lateral ventricle. Several transient or more long-lasting "improvements" were noted in the pilot case. These involved increases of blood flow and nicotine binding as evaluated by positron-emission tomography as well as improvement of the EEG and certain psychological tests, tapping verbal episodic memory. Negative effects of NGF or formation of antibodies against NGF were not noted. In discussing the pilot case, the one conclusion which appears warranted is that it is reasonable to continue the clinical research with NGF treatment of a low number of patients with Alzheimer's disease. The question of whether neurotrophin-mediated mechanisms are disturbed in Alzheimer's disease is discussed. While this issue cannot be settled at present, it is argued that NGF can be used as a pharmacological agent, whether or not there are any primary disturbances of neurotrophin-mediated mechanisms in Alzheimer's disease. Finally, the possibility that NGF might have other positive or negative effects is discussed. In particular, the increase in blood flow noted in the Alzheimer patient suggests that data from clinical research may also generate a feedback to basic science, thus aiding in attempts to find new treatment strategies for neurodegenerative diseases.
RF- 109

2050798036

Source: https://www.industrydocuments.ucsf.edu/docs/hlgl0000

31

AN- <DIALOG> 08794547
TI- Nerve growth factor pharmacology: application to the treatment of cholinergic neurodegeneration in Alzheimer's disease.
AU- Lapchak PA
SO- <JN> Exp Neurol
CP- UNITED STATES
SO- <PY> Nov 1993
SO- <VO> 124 (1) p16-20
SN- 0014-4886
LA- ENGLISH
DT- JOURNAL ARTICLE REVIEW REVIEW, TUTORIAL
AB- The proposal that NGF or compounds that induce the expression of endogenous NGF may be useful in the treatment of AD is based upon a wealth of evidence showing that NGF effectively attenuates lesion-induced cholinergic deficits and cognitive impairments in animal models. In addition, a recent clinical study with chronic NGF treatment in an AD patient showed promise. Olson et al. showed that NGF treatment increased blood flow, [11C]nicotine uptake, and 11C binding in the cerebral cortex. In addition, NGF infusions normalized EEG patterns and improved performance in word recognition tests. Further clinical trials with either NGF or NGF-enhancing compounds will allow determination of whether the animal-based study approach to designing clinically relevant drugs will be advantageous to the treatment of AD. Further findings on the effectiveness of NGF in AD patients are reported by Lars Olson (this volume).
RF- 48

2050798037

Source: https://www.industrydocuments.ucsf.edu/docs/hlgl0000

32

AN- <DIALOG> 08792438
TI- Brief presentation of the story and present status of studies of the vertebrate cholinergic system.
AU- Karczmar AG
CS- Research Services, Hines VA Hospital, Illinois 60144.
SO- <JN> Neuropsychopharmacology
CP- UNITED STATES
SO- <PY> Nov 1993
SO- <VO> 9 (3) p181-99
SN- 0893-133X
LA- ENGLISH
DT- JOURNAL ARTICLE REVIEW REVIEW, ACADEMIC
AB- This year marks the seventieth anniversary of Otto Loewi's demonstration of chemical transmission generally and autonomic cholinergic transmission specifically and the fortieth anniversary of John Eccles's proof of the existence of central cholinergic transmission. Following these epochal findings, the subsequent studies of the cholinergic system led to discoveries of similarly important phenomena. This review concerns these phenomena, including chemical structure and molecular biology of cholinergic receptors; electrophysiologic and ionic aspects of pre- and postsynaptic cholinergic events; the quantal expression of cholinergic postsynaptic events and activities of their subunits, the elementary events; second messengers and G proteins; synthesis, storage and release of acetylcholine; cholinesterases, anticholinesterases, and war gases; central cholinergic pathways; central cholinergic functions, behaviors, cholinergic EEG and REM sleep; cholinergic ontogeny and teratology; trophic phenomena; and the clinical aspects of the cholinergic system. This review refers to the history as well as the present status of each of these phenomena; furthermore, it describes briefly the nineteenth-century work with calabar bean, pilocarpine, muscarine, and nicotine, that is, the work performed before the promulgation of the cholinergic era.
RF- 171

2050798038

Source: https://www.industrydocuments.ucsf.edu/docs/hlgl0000

33

AN- <DIALOG> 07945705
TI- Basic and clinical psychopharmacology of nicotine.
AU- Le Houezec J Benowitz NL
CS- Department of Medicine, San Francisco General Hospital Medical Center, University of California.
SO- <JN> Clin Chest Med
CP- UNITED STATES
SO- <PY> Dec 1991
SO- <VO> 12 (4) p681-99
SN- 0272-5231
LA- ENGLISH
DT- JOURNAL ARTICLE REVIEW REVIEW, ACADEMIC
AB- Nicotine acts on nearly every physiological system of the human body. While the actions of nicotine on the autonomic nervous system may be of interest in understanding possible deleterious long-term effects of smoking on the cardiovascular system, the actions of nicotine on the central nervous system are of the most interest in understanding why people smoke. Many studies of the effects of nicotine on human cognitive function have been performed. Improvement in attention, learning, reaction time, and problem solving have been reported. However, most of the results are inconclusive owing to methodologic problems. Whether the enhanced performance observed after smoking is attributable to relief of symptoms of abstinence or to a primary effect of nicotine on the brain is not clear. The pharmacodynamic effects of nicotine must be considered in the design of studies of the effects of nicotine on human performance. It is not known if the effects of nicotine on performance are subject to tolerance. The facilitation of performance, perceived as a reinforcement, may tend to lessen throughout the day, as do other effects of nicotine. Studies of chronic tolerance to the behavioral effects of nicotine are needed; comparisons between regular smokers and occasional smokers may be helpful. At present, the facilitatory actions of nicotine on human performance have been explained in terms of arousal. However, nicotine actions cannot be explained in terms of this single concept. Different processes, including attention, stimulus evaluation, and response selection, appear to be involved in the effect of nicotine on human information processing. Finally, one must consider that the predominant effects of nicotine may differ among individuals, as different people smoke for different reasons, and the motives for (rewards of) smoking may vary in different situations.
RF- 122

2050798039

10A04

Source: https://www.industrydocuments.ucsf.edu/docs/hlgl0000

34

AN- <DIALOG> 07797327
TI- Neurophysiological aspects of smoking behaviour: a neuroelectric perspective.
AU- Knott VJ
CS- Clinical Neurophysiological Services, University of Ottawa, Royal Ottawa Hospital, Ontario, Canada.
SO- <JN> Br J Addict
CP- ENGLAND
SO- <PY> May 1991
SO- <VO> 86 (5) p511-5
SN- 0952-0481
LA- ENGLISH
DT- JOURNAL ARTICLE REVIEW REVIEW, TUTORIAL
AB- Many of the current neurobiological hypotheses of tobacco smoking concern relationships between nicotine-induced neurotransmitter alterations--events at the level of the individual neuron--and mental behavioural states. Since non-invasive physiological variables may reflect neural activity more directly than overt indices, neuroelectric studies are suggested to be particularly helpful in understanding the intermediate steps between the neuronal effects of smoke/nicotine and behavioural change. The intent of this paper is to offer direction and suggestion regarding electroencephalographic (EEG) and event-related brain potential (ERP) research into smoke/nicotine effects on mood and performance. Suggested directions revolve around multi-component measurement strategies and the employment of study designs and test paradigms which may offer a more dynamic and detailed neurobiological view of smoke/nicotine effects on these processes.
RF- 23

35

AN- <DIALOG> 07506097
TI- Dopaminergic mechanisms in the locomotor stimulant effects of nicotine.
AU- Clarke PB
CS- Department of Pharmacology and Therapeutics, McGill University, Montreal, Canada.
SO- <JN> Biochem Pharmacol
CP- ENGLAND
SO- <PY> Oct 1 1990
SO- <VO> 40 (7) p1427-32
SN- 0006-2952
LA- ENGLISH
DT- JOURNAL ARTICLE REVIEW REVIEW, TUTORIAL
RF- 49

2050798040

Source: https://www.industrydocuments.ucsf.edu/docs/hlgl0000

36

AN- <DIALOG> 07488567
TI- Pharmacology of noradrenaline and neuropeptide tyrosine (NPY)-mediated sympathetic cotransmission.
AU- Lundberg JM Franco-Cereceda A Hemsen A Lacroix JS Pernow J
CS- Department of Pharmacology, Karolinska Institute, Stockholm, Sweden.
SO- <JN> Fundam Clin Pharmacol
CP- FRANCE
SO- <PY> 1990
SO- <VO> 4 (4) p373-91
SN- 0767-3981
LA- ENGLISH
DT- JOURNAL ARTICLE REVIEW REVIEW, TUTORIAL
AB- Pharmacological and physiological aspects for neuropeptide Y (NPY) and noradrenaline (NA) cotransmission have been studied in the peripheral sympathetic nervous control of blood vessels, heart, spleen and vas deferens. NPY coexists with NA in large dense cored vesicles and is released compared to NA mainly upon high frequency stimulation or strong reflex sympathetic activation. NPY release is inhibited via prejunctional alpha-2 adrenoceptors and adenosine receptors but facilitated by angiotensin II or beta-receptor activation. NPY exerts prejunctional inhibitory actions on both NA and NPY release, enhances the vasoconstrictor effect of NA and evokes potent, long-lasting vasoconstriction. Specific receptor mechanisms for NPY exist at both the pre- and postjunctional levels; a large amidated C-terminal portion of NPY is necessary for receptor binding, inhibition of cyclic AMP formation and vasoconstrictor effects. Denervation results in supersensitivity for both NA and NPY-evoked vasoconstriction. Reserpine pretreatment is associated with depletion of NA as well as NPY; the effect on NPY is entirely dependent on an intact nerve activity. Reserpine treatment combined with preganglionic denervation depletes NA by 99% while NPY levels are maintained intact. The characteristic appearance of the nerve stimulation evoked vasoconstrictor response with a high correlation to NPY outflow after reserpine treatment, suggests that NPY may be involved as a transmitter in a variety of vascular beds. NPY-synthesis in ganglia seems to be regulated by nicotinic receptor activity; secondary stimulation by eg reserpine stimulates and nicotine antagonists decrease NPY-synthesis. Many classical pharmacological agents including guanethidine, clonidine, yohimbine, angiotensin II, nicotine and desipramine influence NPY release. A complex interplay therefore seems to occur at both the pre- and postjunctional levels of transmission for the classical transmitter NA and the coexisting peptide NPY, creating a great diversity of chemical signalling potential.
RF- 82

2050298041

Source: https://www.industrydocuments.ucsf.edu/docs/hlgl0000

37

AN- <DIALOG> 06883346
TI- Control of release of vasopressin by neuroendocrine reflexes.
AU- Bisset GW Chowdrey HS
CS- Division of Neurophysiology and Neuropharmacology, National Institute for Medical Research, Mill Hill, London.
SO- <JN> Q J Exp Physiol
CP- ENGLAND
SO- <PY> Nov 1988
SO- <VO> 73 (6) p811-72
SN- 0144-8757
LA- ENGLISH
DT- JOURNAL ARTICLE REVIEW REVIEW, ACADEMIC
AB- The neurones in the supraoptic and paraventricular nuclei (SON and PVN) which secrete vasopressin are separate from those which secrete oxytocin and are distributed in different parts of the nuclei. They may be distinguished electrophysiologically by a characteristic phasic pattern of firing. A selective afferent neural input to these neurones would provide a mechanism for the release of vasopressin independently of oxytocin in response to appropriate physiological stimuli. Release of vasopressin is controlled by changes in blood volume or pressure ('volume control') and in plasma osmolality ('osmotic control'). Stimuli involved in volume control such as haemorrhage, hypotension and carotid occlusion cause vasopressin to be released into the circulation with little or no detectable oxytocin. An osmotic stimulus releases vasopressin alone in some species but not apparently in the rat in which both hormones are released. Volume control is mediated reflexly by peripheral receptors in the cardiovascular system. Activation of baro- and stretch receptors results in inhibition, and activation of chemoreceptors in stimulation, of release. Afferent impulses from these receptors are conveyed in the vagi and carotid sinus nerves to the NTS on the dorsal surface of the brain stem. All afferent impulses to the NTS are excitatory. It follows that the afferents from chemoreceptors must stimulate an excitatory, and those from baro- and stretch receptors an inhibitory, projection from the NTS to the vasopressin-secreting cells in the SON and PVN. Two alternative models are presented of the neural pathways and transmitters involved. The model of Fig. 2 shows an excitatory relay through a cholinoceptive area on the ventral surface of the brain stem which has been termed the 'nicotine-sensitive area' because topical application of nicotine to this area in the cat released vasopressin without oxytocin. An inhibitory relay is shown through the A1 group of noradrenergic neurones on the ventral surface which selectively innervate the vasopressin-secreting neurones in the SON. This model implies an inhibitory role for noradrenaline acting on beta- or alpha 2-receptors. However the most recent investigations suggest an excitatory, rather than inhibitory, function of the A1 noradrenergic neurones involving alpha 1-receptors. This is the basis of the model in Fig. 3. The A1 neurones project either directly to the SON and PVN or indirectly through the lateral preoptic nucleus which lies in close proximity to the SON. The nicotine-sensitive area may be coincident with the A1 group of noradrenergic neurones.(ABSTRACT TRUNCATED AT 400 WORDS)
RF- 480

2050298042

10A04

Source: https://www.industrydocuments.ucsf.edu/docs/hlgl0000

38

AN- <DIALOG> 06369468
TI- Classification of opioid and 5-hydroxytryptamine receptors by means of discriminative drug effects.
AU- Stolerman IP Stephenson JD Rauch RJ
CS- Department of Pharmacology, Institute of Psychiatry, De Crespigny Park, London, U.K.
SO- <JN> Neuropharmacology
CP- ENGLAND
SO- <PY> Jul 1987
SO- <VO> 26 (7B) p867-75
SN- 0028-3908
LA- ENGLISH
DT- JOURNAL ARTICLE REVIEW REVIEW, TUTORIAL
AB- Behavioural studies can help to validate, modify and refine schemes for classifying receptors that are developed from electrophysiological and biochemical experiments. Drug discrimination constitutes one family of behavioural techniques that is being extensively used for studying subtypes of receptors, mainly because the methods often have remarkably high pharmacological specificity but can be applied to agents from a diverse range of classes. This article reviews briefly studies on agents acting through opioid and 5-hydroxytryptamine systems, where the results of the behavioural studies are very largely consistent with findings from other approaches. Many drugs used in such work have limited selectivity for putative subtypes of receptor, but little is known about how such compound pharmacological stimuli are processed in drug discrimination experiments. The characteristics of the discriminative stimuli produced by a mixture of drugs are discussed with respect to implications for effects of single drugs with multiple actions. Based on these initial experiments on discrimination of a mixture of nicotine and midazolam, it appears that the components of a compound pharmacological stimulus may be perceived and processed independently.
RF- 63

39

AN- <DIALOG> 04988970
TI- Smoking, nicotine and electrocortical activity.
AU- Edwards JA Warburton DM
SO- <JN> Pharmacol Ther
CP- ENGLAND
SO- <PY> 1982
SO- <VO> 19 (2) p147-64
SN- 0163-7258
LA- ENGLISH
DT- JOURNAL ARTICLE REVIEW
RF- 76

2050780043

Source: https://www.industrydocuments.ucsf.edu/docs/hlgl0000

40

AN- <DIALOG> 02989371
TI- Neurophysiological and behavioral consequences of chronic nicotine treatment. pp. 375-86.
AU- Nelsen JM
SO- <JN> In: Singh JM, Lal H, ed. Drug addiction Vol 3: Neurobiology and influences on behavior. New York, Stratton, 1974. W3 IN91KC 1973d.
CP- UNITED STATES
CA- W3 IN197KC 1973d
LA- ENGLISH
DT- MONOGRAPH

41

AN- <DIALOG> 02932618
TI- Psychoactive drugs and anxiety, their influence on cerebral circulation and metabolism. A round table discussion. pp. 455-77.
AU- Rafaelsen OJ
SO- <JN> In: Ingvar DH, Lassen NA, ed. Brain work: the coupling of function metabolism and blood flow in the brain. Copenhagen, Munksgaard, 1975. W3 AL36 1974b.
CP- ITALY
CA- W3 AL36 1974b
LA- ENGLISH
DT- MONOGRAPH REVIEW
RF- 21

42

AN- <DIALOG> 02103491
TI- Pharmacology of the brain: the hippocampus, learning and seizures.
AU- Izquierdo I Nasello AG
SO- <JN> Prog Drug Res
CP- SWITZERLAND
SO- <PY> 1972
SO- <VO> 16 p211-28
SN- 0071-786X
LA- ENGLISH
DT- JOURNAL ARTICLE REVIEW
RF- 134

2050798044

Source: https://www.industrydocuments.ucsf.edu/docs/hlgl0000

43

AN- <DIALOG> 02036533
TI- Hippocampal physiology: experiments on regulation of its electrical activity, on the mechanism of seizures, and on a hypothesis of learning.
AU- Izquierdo I
SO- <JN> Behav Biol
CP- UNITED STATES
SO- <PY> Oct 1972
SO- <VO> 7 (5) p669-98
SN- 0091-6773
LA- ENGLISH
DT- JOURNAL ARTICLE REVIEW
RF- 151

44

AN- <DIALOG> 01655190
TI- The dependence of hippocampal function on extracellular potassium levels.
AU- Izquierdo I Nasello AG Marichich ES
SO- <JN> Curr Mod Biol
CP- NETHERLANDS
SO- <PY> May 1971
SO- <VO> 4 (1) p35-46
SN- 0011-4014
LA- ENGLISH
DT- JOURNAL ARTICLE REVIEW
RF- 113

45

AN- <DIALOG> 01533303
TI- [Neuropharmacological approach to sleep]
AU- Yamamoto K
SO- <JN> Saishin Igaku
CP- JAPAN
SO- <PY> Jan 1971
SO- <VO> 26 (1) p16-34
LA- JAPANESE
DT- JOURNAL ARTICLE REVIEW
RF- 158

Source: https://www.industrydocuments.ucsf.edu/docs/hlgl0000

46

AN- <DIALOG> 01494903
TI- Transmission in sympathetic ganglia.
AU- Volle RL Hancock JC
SO- <JN> Fed Proc
CP- UNITED STATES
SO- <PY> Nov-Dec 1970
SO- <VO> 29 (6) p1913-8
SN- 0014-9446
LA- ENGLISH
DT- JOURNAL ARTICLE REVIEW
RF- 40

47

AN- <DIALOG> 01391612
TI- Vascular smooth muscle. II. Pharmacology of normal and hypotensive vessels.
AU- Somlyo AP Somlyo AV
SO- <JN> Pharmacol Rev
CP- UNITED STATES
SO- <PY> Jun 1970
SO- <VO> 22 (2) p249-353
SN- 0031-6997
LA- ENGLISH
DT- JOURNAL ARTICLE REVIEW
RF- 995

48

AN- <DIALOG> 01306712
TI- [Psychopharmacology of learning and memory]
TI- <Original> Psychofarmakologie uceni a pameti.
AU- Bohdanecky Z
SO- <JN> Act Nerv Super (Praha)
CP- CZECHOSLOVAKIA
SO- <PY> Jan 12 1970
SO- <VO> 12 (1) p22-34
LA- CZECH
DT- JOURNAL ARTICLE REVIEW
RF- 74

Source: https://www.industrydocuments.ucsf.edu/docs/hlgl0000

49

AN- <DIALOG> 01215798
TI- [Parasympathetic innervation of the intestine]
TI- <Original> Parasimpaticheskaia innervatsiia kischechnika.
AU- Korotkov AG
SO- <JN> Arkh Anat Gistol Embriol
CP- USSR
SO- <PY> Jun 1969
SO- <VO> 56 (6) p92-109
SN- 0004-1947
LA- RUSSIAN
DT- JOURNAL ARTICLE REVIEW
RF- 102

50

AN- <DIALOG> 01098276
TI- Ureteral motility.
AU- Boyarsky S Labay P
SO- <JN> Annu Rev Med
CP- UNITED STATES
SO- <PY> 1969
SO- <VO> 20 p383-94
SN- 0066-4219
LA- ENGLISH
DT- JOURNAL ARTICLE REVIEW
RF- 96

2050798047

Source: https://www.industrydocuments.ucsf.edu/docs/hlgl0000

FROM SCISEARCH {ISI's *Science Citation Index*; DIALOG: File 434}

51

GA- NW085
NR- 159
TI- IS SCHIZOPHRENIA DUE TO EXCESSIVE SYNAPTIC PRUNING IN THE PREFRONTAL CORTEX - THE FEINBERG HYPOTHESIS REVISITED
AU- KESHAVAN MS ANDERSON S PETTEGREW JW
CS- UNIV PITTSBURGH,WESTERN PSYCHIAT INST & CLIN,DEPT PSYCHIAT,3811 OHARA ST/PITTSBURGH//PA/15213
SO- <JN> JOURNAL OF PSYCHIATRIC RESEARCH
SO- <PY> 1994
SO- <VO> 28
SO- <IS> N3
SO- <IS> MAY-JUN
SO- <PG> 239-265
SN- 0022-3956
LA- ENGLISH
DT- REVIEW
AB- Several lines of evidence support the notion that a substantial reorganization of cortical connections, involving a programmed synaptic pruning, takes place during adolescence in humans. A review of neurobiological abnormalities in schizophrenia indicates that the neurobiological parameters that undergo peripubertal regressive changes may be abnormal in this disorder. An excessive pruning of the prefrontal corticocortical, and corticosubcortical synapses, perhaps involving the excitatory glutamatergic inputs to pyramidal neurons, may underlie schizophrenia. A reciprocal failure of pruning in certain subcortical structures, such as lenticular nuclei, may also occur. Several developmental trajectories, related to early brain insults as well as genetic factors affecting postnatal neurodevelopment, could lead to the illness. These models would have heuristic value and may be consistent with several known facts of the schizophrenic illness; such as its onset in adolescence and the gender differences in its onset and natural course. The relationship between these models and other etiological models of schizophrenia are summarized and approaches to teal relevant hypotheses are discussed.

2050798048

Source: https://www.industrydocuments.ucsf.edu/docs/hlgl0000

52

GA- HY865
NR- 34
TI- EFFECTS OF TOBACCO SMOKING ON THE TOPOGRAPHIC EEG .2.
AU- DOMINO EF RISKALLA M ZHANG YF KIM E
CS- UNIV MICHIGAN,DEPT PHARMACOL,6322 MED SCI BLDG/ANN ARBOR//MI/48109
SO- <JN> PROGRESS IN NEURO-PSYCHOPHARMACOLOGY & BIOLOGICAL PSYCHIATRY
SO- <PY> 1992
SO- <VO> 16
SO- <IS> N4
SO- <IS> JUL
SO- <PG> 463-482
LA- ENGLISH
DT- REVIEW
AB- 1. Tobacco smokers are well aware of the long term hazards of tobacco smoking, yet they continue to smoke. Presumably people smoke because of short term gains due to nicotine. 2. The mechanism by which nicotine is a drug reinforcer still needs a great deal of study. The specific aim of the present study was to determine the effects of tobacco smoki.Ke 9 the topographic EEG of 12 hr deprived heavy tobacco smokers. 3. Seven normal adult tobacco smokers of mixed sex were recruited into the study and compared with six normal nonsmokers of similar age and sex. 4. A Grass Model 8-24D EEG and 16 different scalp monopolar electrodes were used to record the EEG using both ears as reference before and after smoking. EKG lead 11 was recorded on channel 17. Blood pressure was measured by auscultation. Exhaled CO was measured using a CO detector. Computer analysis of the EEG data was run off line on a Zenith 386/25 microcomputer using RHYTHM 7.1. The same system was used to store the EEG in digitized form. The maximum number of 4 sec artifact free epochs in a 3 min recording period with eyes open and then closed was used before and after low and high nicotine tobacco or sham smoking. 5. The hypothesis of this research was confirmed, i.e., that tobacco smoking of high nicotine cigarettes (about 2.0 mg/cigarette) would cause a shift in EEG alpha rhythm to higher frequencies in more diffuse midline cortical structures. In other studies an increase in alpha rhythm has been correlated with an awake relaxed behavioral state. 6. A heart rate increase was a more sensitive index of tobacco smoking than an increase in arterial blood pressure. Exhaled smoking CO levels correlated the nicotine and tar content of the cigarette.

2050298049

Source: https://www.industrydocuments.ucsf.edu/docs/hlgl0000

SMOKING/CIGS, EP/ERP/EEG ETC. & HEALTHY/NORMAL VOLS/SUBJECTS

FROM EMBASE {*Excerpta Medica*; DIALOG: File 73}

53

AN- <DIALOG> 8033392
TI- Vibration perception threshold: Influence of age, height, sex, and smoking, and calculation of accurate centile values
AU- Wiles P.G. Pearce S.M. Rice P.J.S. Mitchell J.M.O.
CS- University Department of Medicine, The General Infirmary, Leeds LS1 3EX
CS- United Kingdom
SO- <JN> DIABETIC MED.
CP- United Kingdom
SO- <PY> 1991
SO- <VO> 8/2 (157-161)
SN- 0742-3071
LA- English
AB- Vibration perception threshold (VPT) is increasingly used as a measure of large nerve fibre function in studies of patients with diabetes and in other disorders. In order to establish the influence of age, height, sex, and smoking on VPT values in the normal population and to allow the calculation of accurate age-related percentile charts, 1,365 healthy volunteers were studied using a biothesiometer. Measurements were made bilaterally on thumbs, great toes, and over medial malleoli. Multivariate regression analysis confirmed age to be the major determinant of VPT levels at all sites ($p < 0.001$). Height was a significant factor for toes and ankles ($p < 0.001$) but not thumbs. Sex had no overall effect at toe or thumb but there were differences regarding ankle VPT ($p < 0.01$). Log transformation of VPT data produced a linear relationship with age at all sites except at the thumbs in elderly females where there was significant deviation from this model ($p < 0.001$) and inverse square root transformation was more appropriate. Smoking had no effect on VPT levels. Age-related centile charts were produced for each site and an easy-to-use computer program was developed to calculate centile values based on raw VPT data, age, height, and sex.

2050298050

Source: https://www.industrydocuments.ucsf.edu/docs/hlgl0000

54

AN- <DIALOG> 6313553
TI- Evoked taste thresholds in a normal population and the application of electrogustometry to trigeminal nerve disease
AU- Grant R. Ferguson M.M. Strang R. et al.
CS- Department of Neurology, Southern General Hospital, Glasgow G51 4TF
CS- UNITED KINGDOM
SO- <JN> J. NEUROL. NEUROSURG. PSYCHIATRY
CP- UK
SO- <PY> 1987
SO- <VO> 50/1 (12-21)
LA- ENGLISH
AB- No standardised method for taste threshold measurement is available and therefore comparison between clinical studies is difficult. An electrogustometer was evaluated in normal subjects. No sex difference in test threshold was noted; however, there was a significant elevation in detection thresholds in age and smoking. Electrogustometric values both in patients before and after surgery for trigeminal neuralgia and in patients with trigeminal sensory neuropathy were determined. Many patients with trigeminal nerve disorders had abnormal electrogustometric detection thresholds suggesting that there is possibly an accessory taste pathway through the trigeminal nerve, although in some individuals the site of lesion may be in the brain stem. Electrogustometry is a convenient method for clinically assessing taste.

55

AN- <DIALOG> 6293465
TI- Transient pattern-evoked potentials in heavy smokers
TI- <ORIGINAL> ZUM VERHALTEN VISUELL EVOZIERTER POTENTIALE (MUSTER-UMKEHR-VECP) BEI RAUCHERN
AU- Fotzsch R. Podemski R. Brzecki A. Bogdanska R.
CS- Klinik fur Psychiatrie und Neurologie der Medizinischen Akademie 'Carl Gustav Carus', DDR-8091 Dresden
CS- GERMANY, EAST
SO- <JN> PSYCHIATR. NEUROL. MED. PSYCHOL.
CP- GERMANY, EAST
SO- <PY> 1986
SO- <VO> 38/9 (518-525)
LA- GERMAN
SL- RUSSIAN ENGLISH
AB- Twenty six clinically healthy smokers were studied with transient pattern visual-evoked potentials. In comparison with a sample of 'normal' subjects we found a difference of some parameters ($p<0.05$): the Psub 2-latency and the interocular difference of latency on average were 6-7 ms respectively 2 ms larger. Thirteen subjects have had a prolonged Psub 2-latency ($P<0.05$) and/or a difference in interocular latencies larger than 5 ms. These findings may suggest on subclinical disturbances of visual system and indicators of an especial sensitivity of that persons. The findings are possibly related to tobacco-alcohol amblyopia.

2050298051

Source: https://www.industrydocuments.ucsf.edu/docs/hlgl0000

56

AN- <DIALOG> 5162891
TI- Clinical ERG findings in tobacco-alcohol amblyopia
AU- Hennekes R.
CS- Univ. Augenklin., D-4000 Dusseldorf
CS- GERMANY, WEST
SO- <JN> GRAEFE'S ARCH. CLIN. EXP. OPHTHALMOL.
CP- GERMANY, WEST
SO- <PY> 1982
SO- <VO> 219/1 (38-39)
LA- ENGLISH
AB- Eight patients with tobacco-alcohol amblyopia, but no significant changes in ERG configuration, were subjected to a more extended ERG analysis applying normalized a- and b-wave amplitude-intensity relationships. The functions established significantly fell out of the range of normal subjects, with respect to the maximum of the b-wave function in dark and light adaptation. In this way, the ERG discrimination level in detecting retinal disturbances in tobacco-alcohol amblyopia can be shifted towards higher sensitivity.

FROM MEDLINE {DIALOG: FILE 155}

57

AN- <DIALOG> 06118780
TI- [Behavior of visual evoked potentials (pattern reversal VECP) in smokers]
TI- <Original> Zum Verhalten visuell evozierter Potentiale (Muster-Umkehr-VECP) bei Rauchern.
AU- Fotzsch R Podemski R Brzecki A Bogdanska R
SO- <JN> Psychiatr Neurol Med Psychol (Leipz)
CP- GERMANY, EAST
SO- <PY> Sep 1986
SO- <VO> 38 (9) p518-25
SN- 0033-2739
LA- GERMAN
SL- ENGLISH
DT- JOURNAL ARTICLE
DT- English Abstract
AB- Twenty six clinically healthy smokers were studied with transient pattern visual-evoked potentials. In comparison with a sample of "normal" subjects we found a difference of some parameters (P less than 0.05): the P2-latency and the interocular difference of latency on average were 6-7 ms respectively 2 ms larger. Thirteen subjects have had a prolonged P2-latency (P less than 0.05) and/or a difference in interocular latencies larger than 5 ms. These findings may suggest on subclinical disturbances of visual system and indicators of an special sensitivity of that person. The findings are possibly related to tobacco-alcohol amblyopia.

2050798052

Source: https://www.industrydocuments.ucsf.edu/docs/hlgl0000

FROM SCISEARCH {ISI's *Science Citation Index*; DIALOG: File 434}

58

GA- AP582
NR- 11
TI- EFFECT OF CIGARETTE-SMOKING ON THE FREQUENCY OF VENTRICULAR PREMATURE COMPLEXES IN NORMAL SUBJECTS
AU- PESTELL RG TAYLOR RR
CS- ROYAL PERTH HOSP,DEPT CARDIOL,VICTORIA SQ/PERTH/WA 6000/AUSTRALIA/ ROYAL PERTH HOSP,DEPT CARDIOL,VICTORIA SQ/PERTH/WA 6000/AUSTRALIA/ ROYAL PERTH HOSP,DEPT MED/PERTH/WA 6001/AUSTRALIA/ UNIV WESTERN AUSTRALIA/PERTH/WA/AUSTRALIA/
SO- <JN> CLINICAL AND EXPERIMENTAL PHARMACOLOGY & PHYSIOLOGY
SO- <PY> 1989
SO- <VO> 16
SO- <IS> N8
SO- <PG> 647-650
LA- ENGLISH
DT- ARTICLE

2050798053

Source: https://www.industrydocuments.ucsf.edu/docs/hlgl0000

SMOKING, EP/EEG/ERP ETC., & REVS, BOOK CHAPTERS, MONOGRAPHS, ETC.

FROM psycINFO {DIALOG: FILE 11}

59

AN- 00142022
TI- Additional characteristic EEG differences between smokers and nonsmokers.
AU- Brown, Barbara B.
CS- Veterans Administration Hosp., Sepulveda, Calif.
SO- In W. L. Dunn (Ed.), Smoking behavior: Motives and incentives. Washington, D.C.: V. H. Winston & Sons, 1973. xii, 309 p. $9.95
LA- English
DT- BOOK CHAPTER
AB- Analyzed EEG records and personality characteristics of smokers and nonsmokers. Results are compared to previous studies relating smoking to EEG synchronizing mechanisms, personality correlates, and the action of sedative drugs. (67 ref.)
AB- (PsycINFO Database Copyright 1973 American Psychological Assn, all rights reserved)

FROM EMBASE {*Excerpta Medica*; DIALOG: File 73}

60

AN- <DIALOG> 909006
TI- Drugs and breast feeding: a review
AU- Anderson P.O.
CS- Dept. Pharmaceut. Serv., Univ. Hosp., Univ. California Med. Cent., San Diego, Calif. 92103
CS- USA
SO- <JN> DRUG INTELLIGENCE CLIN.PHARM.
CP- USA
SO- <PY> 1977
SO- <VO> 11/4 (208-223)
LA- ENGLISH

2050798054

Source: https://www.industrydocuments.ucsf.edu/docs/hlgl0000

FROM MEDLINE {DIALOG: FILE 155}

61

AN- <DIALOG> 08036351
TI- Cigarette smoking and the adrenergic nervous system.
AU- Grassi G Seravalle G Calhoun DA Bolla G Mancia G
CS- Cattedra di Semeiotica Medica, Universita di Milano, Italy.
SO- <JN> Clin Exp Hypertens [A]
CP- UNITED STATES
SO- <PY> 1992
SO- <VO> 14 (1-2) p251-60
SN- 0730-0077
LA- ENGLISH
DT- JOURNAL ARTICLE REVIEW REVIEW, TUTORIAL
AB- The acute increase in blood pressure and heart rate that accompanies cigarette smoking is associated with a rise in plasma catecholamines and it is thus believed to result from stimulation of the adrenergic nervous system. We have employed direct recording of efferent post-ganglionic sympathetic nerve activity by the microneurographic technique from the peroneal nerve to determine whether this stimulation occurs centrally or peripherally. It was shown that during cigarette smoking blood pressure, heart rate, plasma norepinephrine and epinephrine do increase markedly. Sympathetic nerve activity, however, shows a concomitant specular reduction. Thus peripheral (adrenal gland stimulation, reduction in norepinephrine reuptake, reduction in catecholamine clearance, etc.) rather than central mechanisms explain the adrenergic involvement in the acute hemodynamic effgCh.....K4smoking, the central sympathetic drive being inhibited rather than excited probably as a result of arterial baroreceptor stimulation.
RF- 12

2050798055

Source: https://www.industrydocuments.ucsf.edu/docs/hlgl0000

62

AN- <DIALOG> 07160530
TI- Leber's hereditary optic atrophy.
AU- Berninger TA Bird AC Arden GB
CS- University Eye Hospital Munich, West Germany.
SO- <JN> Ophthalmic Paediatr Genet
CP- NETHERLANDS
SO- <PY> Sep 1989
SO- <VO> 10 (3) p211-27
SN- 0167-6784
LA- ENGLISH
DT- JOURNAL ARTICLE REVIEW REVIEW, TUTORIAL
AB- Leber described a particular type of hereditary optic atrophy in 1871. The clinical features of all cases since reported are reviewed. There is characteristically acute visual loss, circumpapillary teleangiectatic micropathy, tortuosity of the retinal vessels and oedema in the retinal nerve fibres. 85% of those affected are male, but affected fathers do not transmit the condition to their children. The exact mode of inheritance is still obscure but it is suggested that inheritance may be mitochondrial; enlarged subsarcolemmal mitochondria in LOA patients have been described. Colour vision defects are observed not only in patients, but also in presumed carriers. Electrophysiological investigations demonstrate optic nerve damage, but are not indicative of any particular pathology. It has been reported that in many cases of LOA the severity of the disease is related to tobacco smoking. Increased cyanocobalamin blood levels in patients and increased cyanide blood levels support this hypothesis.
RF- 88

Source: https://www.industrydocuments.ucsf.edu/docs/hlgl0000

63

AN- <DIALOG> 05346037
TI- How information processing mode could affect prostaglandin E1 metabolism and lung inactivation: relevance of hemispheric specialization, neurotransmitter asymmetry and brain reactivity.
AU- Backon J
SO- <JN> Med Hypotheses
CP- ENGLAND
SO- <PY> Jun 1984
SO- <VO> 14 (2) p199-206
SN- 0306-9877
LA- ENGLISH
DT- JOURNAL ARTICLE REVIEW
AB- Due to asymmetry of brain neurotransmitters and differential hemispheric information processing modes, it is suggested that the excessive use of one information processing mode could engender a state of brain reactivity whose neurochemical correlates would be either a rise in melatonin or beta-endorphin in systemic circulation. Since melatonin and beta-endorphin have opposite effects on lung-mediated regulation of prostaglandins, it is further suggested that the pulmonary inactivation of prostaglandin E1 would either be increased or inhibited. Low levels of PGE1 would engender high levels of PGE2 whose effects would explain the findings in schizophrenics of: 'reducing' pattern of visual evoked response, cerebral atrophy, and viral and autoimmune phenomena. The primacy of the disordered cognitive style in leading up to the immunological, biochemical and neuropathological processes is stressed. Implications of this model for understanding depression, anxiety and phobic disorders, autism, attention deficit disorder, obesity, alcoholism, smoking, drug addiction, sexual deviations, and certain psychosomatic and psychophysiological disorders are suggested.
RF- 67

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Source: https://www.industrydocuments.ucsf.edu/docs/hlgl0000

FROM SCISEARCH {ISI's *Science Citation Index*; DIALOG: File 434}

64

GA- LR727
NR- 157
TI- HUMAN BRAIN MEASURES OF CLINICAL PAIN - A REVIEW .1. TOPOGRAPHIC MAPPINGS
AU- CHEN ACN
CS- NEUROCOGNIT INST,14359 NORDHOFF ST/LOS ANGELES//CA/91402
SO- <JN> PAIN
SO- <PY> 1993
SO- <VO> 54
SO- <IS> N2
SO- <IS> AUG
SO- <PG> 115-132
SN- 0304-3959
LA- ENGLISH
DT- REVIEW
AB- A comprehensive paper (Parts I and II) has been developed to review the cerebral measures employed in studying the brain neurophysiological activities of clinical pain. Part I focuses on the electro-, magnetic-physiological assessment of clinical pain, and Part II concerns the anatomico-, chemical-physiological assessment of clinical pain. In Part I, these measures include the qualitative inspection of the conventional electroencephalogram, quantitative assessment of brain electrical spectral activity through cortical power spectrum density and coherence analyses, and quantitative averaging of cortical electrical or magnetic activities using brain evoked potentials. The mapping and measurement of these electrical activities and magnetic fields are results of recent advent in computer technology and advanced algorithms. Promises and limitations of these topographic measures in understanding pain in the brain are stated. The next article (Part II) of this paper will review tomographic imaging of pain-related brain activities in regional cerebral flow, the scanning of gross and fine brain structures by computerized axial tomography or magnetic resonance imaging, and the imaging and measurement of brain metabolic changes, energy uptake, and receptor bindings through positron emission tomography or single-photon emission computerized tomography. Molecular chemical transformation by the nuclear magnetic resonance analysis of tissue changes and analgesic-receptor interactions will also be noted.

2050798058

Source: https://www.industrydocuments.ucsf.edu/docs/hlgl0000

65

GA- FC716
NR- 32
TI- PERFORMANCE TESTS IN HUMAN PSYCHOPHARMACOLOGY .1. TEST RELIABILITY AND STANDARDIZATION
AU- PARROTT AC
CS- POLYTECH E LONDON,DEPT PSYCHOL/LONDON E15 4LZ//ENGLAND/
SO- <JN> HUMAN PSYCHOPHARMACOLOGY-CLINICAL AND EXPERIMENTAL
SO- <PY> 1991
SO- <VO> 6
SO- <IS> N1
SO- <PG> 1-9
LA- ENGLISH
DT- REVIEW
AB- Reliability, validity and standardization are well-established principles in most areas of psychometrics, but are rarely mentioned in performance assessment within human psychopharmacology. Test reliability and standardization are examined here, while validity is covered in two succeeding articles. The undocumented reliability of human psychopharmacology performance tests makes the interpretation of findings difficult and ambiguous. Reliability, or consistency within and between test sessions is, however, easy to calculate. Several procedures for calculating reliability are described, with test-retest reliability recommended as the most appropriate single summary measure. Poor standardization is another major problem, with many research groups using different tests. The extent of this problem is examined, and a set of standardization requirements proposed. They could comprise the basis for test manuals. The problems of undocumented reliability and unstandardized tests have been recognized within the field for many years, yet no moves have been made to remedy the situation. One simple solution would be for psychopharmacology journals to accept only documented tests, and to request test-retest reliability data.

66

GA- DJ947
NR- 148
TI- A SEARCH FOR EXPLANATION OF THE BRAIN AND LEARNING - ELEMENTS OF THE PSYCHONOMIC INTERFACE BETWEEN PSYCHOLOGY AND NEUROPHYSIOLOGY .1. A COGNITIVE APPROACH TO EARLY LEARNING
AU- BURTON PG
CS- UNIV WOLLONGONG,COGNIT SCI GRP/WOLLONGONG/NSW 2500/AUSTRALIA/
SO- <JN> PSYCHOBIOLOGY
SO- <PY> 1990
SO- <VO> 18
SO- <IS> N2
SO- <PG> 119-161
LA- ENGLISH
DT- REVIEW

2050798059

Source: https://www.industrydocuments.ucsf.edu/docs/hlgl0000

67

GA- T3894
NR- 68
TI- NEUROBEHAVIOURAL ABNORMALITIES IN PARKINSONS-DISEASE AND THEIR RELATIONSHIP TO PSYCHOMOTOR RETARDATION AND OBSESSIONAL COMPULSIVE DISORDERS
AU- LEES AJ
CS- NATL HOSP,QUEENS SQ/LONDON WC1N 3BG//ENGLAND/
SO- <JN> NERVENARZT
SO- <PY> 1989
SO- <VO> 60
SO- <IS> N2
SO- <PG> 71-79
LA- GERMAN
DT- REVIEW

68

GA- H4687
NR- 111
TI- DRUG-INTERACTIONS WITH THE CALCIUM-ENTRY BLOCKERS
AU- PIEPHO RW CULBERTSON VL RHODES RS
CS- UNIV MISSOURI,SCH PHARM,5005 ROCKHILL RD/KANSASCITY//MO/64110 UNIV COLORADO,SCH PHARM/BOULDER//CO/80309
SO- <JN> CIRCULATION
SO- <PY> 1987
SO- <VO> 75
SO- <IS> N6
SO- <PG> 181-194
LA- ENGLISH
DT- REVIEW, BIBLIOGRAPHY

2050798060

Source: https://www.industrydocuments.ucsf.edu/docs/hlgl0000

CAFFEINE & EEG/EP/ERP ETC AND HEALTHY/NORMAL VOLUNTEERS/SUBJECTS
FROM psycINFO {DIALOG: FILE 11}

69

AN- 00517052
TI- Personality traits influence the effects of diazepam and caffeine on CNV magnitude.
AU- Munte, Thomas-Frank Heinze, Hans-Jochen Kunkel, Helmut Scholz, Michael
CS- Medizinische Hochschule Hannover, Inst fur Klinische Neurophysiologie und Experimentelle Neurologie, West Germany
SO- <JN> Neuropsychobiology
SO- 1984 Nov Vol 12(1) 60-67
SN- 0302282X
LA- English
DT- JOURNAL ARTICLE
AB- Investigated the time course of the effects of caffeine (200 mg), diazepam (8 mg), and placebo on contingent negative variation (CNV) magnitude, and state of mood in 14 normal volunteers (aged 20-27 yrs) using a double-foreperiod paradigm. Each S participated in 3 sessions. A CNV recording was made before Ss received medication. Recording was repeated 4 times at hourly intervals. Ss were told to fixate on a dim red light located 2 m in front of them throughout the entire experimental run. Instructions were to terminate a noise burst (60 db) as quickly as possible by pressing a button. Ss then were administered forms that assessed mood changes and personality differences. CNV changes were quantified by means of a linear robust fitting technique. In general, a reduction of amplitude was found for diazepam and an enhancement followed by a suppression in the caffeine condition. Emotional lability of the Ss, as measured by a personality inventory, was shown to strongly influence the nature and time course of these drug effects. Differential drug responses are discussed in terms of the attention arousal model. (19 ref)
AB- (PsycINFO Database Copyright 1985 American Psychological Assn, all rights reserved)

2050798061

Source: https://www.industrydocuments.ucsf.edu/docs/hlgl0000

FROM EMBASE {*Excerpta Medica*; DIALOG: File 73}

70

AN- <DIALOG> 9072277
TI- Effect of caffeine on the recognition of and responses to hypoglycemia in humans
AU- Kerr D. Sherwin R.S. Pavalkis F. Fayad P.B. Sikorski L. Rife F. Tamborlane W.V. During M.J.
CS- Royal Bournemouth Hospital, Castle Lane East, Bournemouth, Dorset BH7 7DW
CS- United Kingdom
SO- <JN> ANN. INTERN. MED.
CP- USA
SO- <PY> 1993
SO- <VO> 119/8 (799-804)
SN- 0003-4819
LA- English
SL- English
AB- Objective: To determine whether two effects of acute caffeine ingestion- decrease in cerebral blood flow and increase in brain glucose use-alter the recognition of and physiologic responses to hypoglycemia. Design: On two occasions, a hyperinsulinemic glucose clamp technique (2 mU/kg body weight per minute) was used to maintain plasma glucose at 5 mmol/L for 90 minutes, followed by 60 minutes at 3.8 mmol/L, and then 2.8 mmol/L. After 30 minutes at 5 mmol/L, participants consumed, using a randomized, double-blind design, caffeine-free cola with or without caffeine (400 mg) added. Setting: Yale Clinical Research Center. Participants: Eighty healthy, nonobese volunteers (5 men; age range, 20 to 33 years). Measurements: Middle cerebral artery velocity (V(MCA)), counter-regulatory hormone levels, hypoglycemic symptoms, and cognitive function (P300 evoked potentials). Results: Caffeine caused an immediate and sustained 23% decrease in V(MCA) from 64 to 49 cm/s (point estimate of difference, +15 cm/s (95% CI, 10 to 21 cm/s), $P < 0.001$). At a glucose level of 3.8 mmol/L, only the participants given caffeine had warning symptoms and 'felt hypoglycemic.' Moreover, the level of epinephrine was 118% ((CI of point difference, 76% to 158%) (CI, $P < 0.001$)) higher after caffeine consumption compared with placebo. Similarly, levels of norepinephrine (41% (CI, 26% to 60%), $P < 0.002$), cortisol (65% (CI, 26% to 78%), $P < 0.008$), and growth hormone (60% (CI, 16% to 143%), $P < 0.05$) were higher after caffeine consumption compared with placebo. At 2.8 mmol/L, epinephrine (40% (point estimate of the percentage difference), $P < 0.05$), norepinephrine (27%, $P < 0.05$), and cortisol (24%, $P < 0.05$) levels were higher, participants were more aware ($P < 0.02$) of hypoglycemia, and P300 latency was prolonged in the group that consumed caffeine (7.2%, $P < 0.05$). Conclusions: Acute ingestion of caffeine is associated with sympathoadrenal activation and awareness of hypoglycemia at a glucose level not usually considered hypoglycemic. Our data suggest that individuals who ingest moderate amounts of caffeine may develop hypoglycemic symptoms if plasma glucose levels fall into the 'low-normal' range, as might occur in the late postprandial period after ingestion of a large carbohydrate load.

2050798062

Source: https://www.industrydocuments.ucsf.edu/docs/hlgl0000

71

AN- <DIALOG> 8670863
TI- Quantitative electroencephalographic effects of caffeine in panic disorder
AU- Newman F. Stein M.B. Trettau J.R. Coppola R. Uhde T.W.
CS- Department of Psychiatry, Grace General Hospital, 300 Booth Drive, Winnipeg, Man. R3J 3M7
CS- Canada
SO- <JN> PSYCHIATRY RES. NEUROIMAGING
CP- Ireland
SO- <PY> 1992
SO- <VO> 45/2 (105-113)
SN- 0925-4927
LA- English
SL- English
AB- It has been demonstrated that patients with panic disorder are more sensitive than normal control subjects to the anxiogenic effects of caffeine. The underlying physiologic basis for this difference is unclear. We examined the electroencephalographic (EEG) activity of seven patients with panic disorder and seven normal control subjects during the randomized double-blind, placebo-controlled administration of oral caffeine (7 mg/kg). EEG data were collected on-line from 28 electrodes; artifact-free epochs were selected manually for off-line Fourier transformation. Caffeine was associated with a significant increase in peak occipital alpha frequency and significant decreases in occipital alpha amplitude, central beta amplitude, and central theta amplitude. Despite the observation that caffeine increased anxiety more in the patients with panic disorder than in the normal control subjects, the two groups did not differ in their EEG responses to caffeine.

2050798063

Source: https://www.industrydocuments.ucsf.edu/docs/hlgl0000

72

AN- <DIALOG> 8661543
TI- Anxiogenic effects of caffeine in patients with anxiety disorders
AU- Bruce M. Scott N. Shine P. Lader M.
CS- Warneford Hospital, Oxford OX3 7JX
CS- United Kingdom
SO- <JN> ARCH. GEN. PSYCHIATRY
CP- USA
SO- <PY> 1992
SO- <VO> 49/11 (867-869)
SN- 0003-990X
LA- English
SL- English
AB- The effects on measures of anxiety from two doses of oral caffeine (250 and 500 mg) and placebo were compared in 12 patients with generalized anxiety disorder (GAD), 12 patients with panic disorder, and 12 normal subjects. Caffeine produced significantly less decrease in electroencephalographic alpha wave activity, greater decrease in N1-P2 auditory evoked potential amplitude, and greater increased in skin conductance level, systolic and diastolic blood pressure, critical fusion flicker frequency, and self-ratings of anxiety and sweating in patients with GAD than in normal patients. Patients with panic disorder showed different reactivity than normal patients did with respect to electroencephalographic alpha waves, N2 latency, N2-P2 auditory evoked potential amplitude, and physical tiredness but were less reactive than patients with GAD on several variables. It is concluded that patients with GAD are abnormally sensitive to caffeine and that the data support the view that panic disorder is a separable disorder from GAD.

Source: https://www.industrydocuments.ucsf.edu/docs/hlgl0000

73

AN- <DIALOG> 7204492
TI- Caffeine and EEG mapping: effects of a visuo-spatial task in healthy volunteers
TI- <ORIGINAL> CAFEINE ET CARTOGRAPHIE EEG: EFFETS D'UNE TACHE VISUOSPATIALE CHEZ DES VOLONTAIRES SAINS. STRATEGIE D'ANALYSE DES DONNEES ELECTROPHARMACOLOGIQUES
AU- Etevenon P. Peron-Magnan P. Guillou S. Toussaint M. Gueguen B Boulenger J.P. Deniker P. Loo H.
CS- Centre Esquirol, CHU, 14033 Caen Cedex
CS- France
SO- <JN> NEUROPHYSIOL. CLIN.
CP- France
SO- <PY> 1988
SO- <VO> 18/4 (355-367)
SN- 0987-7053
LA- French
SL- English
AB- A group of 10 subjects, normal volunteers (6 men, 4 women), mean age 27.2 years, right-handers, has been selected for a double-blind study of caffeine effects (400 mg per os) versus placebo. One week delay separated the two EEG sessions quantified by spectral analysis. During each session (16 EEG channels, common reference), each subject was recorded four times: 2.5 min, in eyes-closed condition, under diffuse attention, followed by 2.5 min, during a visuo-spatial task ('eyes open' condition: presentation of a picture). These two EEG recordings were obtained before the oral administration and repeated 1 hr after the administration of caffeine or placebo. Individual and group results presented globally: 1) an increase in mean frequencies of alpha activity in 'eyes closed' condition, and of delta activity in 'eyes open' condition; 2) a decrease of alpha activity amplitudes (both RMS and % values) in both conditions; 3) a decrease of total RMS amplitudes in 'eyes closed' condition; 4) an increase of relative % amplitudes for beta 2% fast activity in the 'eyes open condition'. These results characterize the psycho-stimulant effects of caffeine, especially over the left temporal area which was confirming an initial statistical hypothesis of specific cerebral local activation. The non-parametric permutation tests of Fisher, were not always reaching statistical significance for the same EEG channel quantified by one of the 17 analyzed spectral parameters, except on the left temporal area. During the visuo-spatial task, the decrease in alpha amplitudes (RMS and %) was also statistically significant, but over larger areas: over left and right temporal, central and parietal regions. In both conditions, eyes closed and eyes open, the alpha RMS amplitude was also decreased over the right anterior frontal area.

Source: https://www.industrydocuments.ucsf.edu/docs/hlgl0000

74

AN- <DIALOG> 7176587
TI- High-dose caffeine and cardiac rate and rhythm in normal subjects
AU- Newcombe P.F. Renton K.W. Rautaharju P.M. Spencer C.A. Montague T.J.
CS- Department of Medicine, Dalhousie University and the Victorial General Hospital, Halifax, NS
CS- Canada
SO- <JN> CHEST
CP- USA
SO- <PY> 1988
SO- <VO> 94/1 (90-94)
SN- 0012-3692
LA- English
AB- In a previous 24-hour study of the electrophysiologic effects of moderate dose caffeine (1 mg/kg body weight/half-life), we found a significant ($p < 0.01$) increase in ventricular ectopic beat (VEB) frequency among 18 patients with preexisting primary ventricular dysrhythm (mean 207 plus or minus 350 VEBs/hour, no caffeine, versus 307 plus or minus 414 VEBs/hour, caffeine). We also found a statistically insignificant (NS) increase in the incidence of infrequent VEBs in 18 normal control subjects (four of 18, no caffeine vs nine of 18 caffeine). Because of the high risk of beta-error among the previously-studied normal control subjects, we tested another group of 34 normal subjects, 15 males and 19 females with a mean age of 31 years (range 21 to 49 years), using a higher dose of caffeine. All subjects abstained from caffeine for 72 hours and had a control 24-hour Holter ECG recorded between hours 48 and 72. Caffeine half-life was calculated for each subject and caffeine was then ingested at 1 mg/kg every 0.5 half-life during all waking hours. A 24-hour Holter test was recorded, beginning just prior to the second caffeine dose. It was concluded that in normal adults, even high-dose caffeine does not affect prevailing cardiac rhythm and rate, and moreover, does not cause clinically significant ventricular or supraventricular dysrhythm.

2050798066

Source: https://www.industrydocuments.ucsf.edu/docs/hlgl0000

75

AN- <DIALOG> 6272438
TI- The psychopharmacological and electrophysiological effects of single doses of caffeine in healthy human subjects
AU- Bruce M. Scott N. Lader M. Marks V.
CS- Department of Pharmacology, Institute of Psychiatry, London SE5 8AF
CS- UNITED KINGDOM
SO- <JN> BR. J. CLIN. PHARMACOL.
CP- UK
SO- <PY> 1986
SO- <VO> 22/1 (81-87)
LA- ENGLISH
AB- 1. The effects of single doses of anhydrous caffeine (250 mg and 500 mg) and placebo on physiological, psychological measures and subjective feelings were studied in a double-blind, cross-over study in nine healthy subjects who had abstained from caffeine-containing beverages for 24 h before each occasion. 2. Caffeine and caffeine metabolites in plasma and urine were assayed. Peak plasma concentrations were observed at 1 to 2 h with an approximate half-life of 5 h. The concentrations of the metabolite 1,7-dimethylxanthine increased during the 5 h. The major urine metabolite was 1-methyluric acid. 3. The EEG showed a dose-related decrease in log 'theta' power and a decrease in log 'alpha' power. Other dose-related effects were an increase in skin conductance level (sweat-gland activity) and self rating of alertness. Ratings of headache and tiredness were decreased by the caffeine. 4. The study illustrates the complexities of studying a drug which is widely taken and which is often associated with withdrawal effects.

2050798067

Source: https://www.industrydocuments.ucsf.edu/docs/hlgl0000

76

AN- <DIALOG> 5844448
TI- The effect of caffeine on cardiac rate, rhythm, and ventricular repolarization. Analysis of 18 normal subjects and 18 patients with primary ventricular dysrhythmia
AU- Sutherland D.J. McPherson D.D. Renton K.W. et al.
CS- Department of Medicine, Dalhousie University, Halifax, N.S., B3H 2Y9
CS- CANADA
SO- <JN> CHEST
CP- USA
SO- <PY> 1985
SO- <VO> 87/3 (319-324)
LA- ENGLISH
AB- To determine clinical electrophysiologic effects of a moderate dose of caffeine, we compared prevailing cardiac rhythm and rate, the prevalence and frequency of ventricular dysrhythmia, and Q-T intervals in two populations over an initial 24-hour caffeine-free period and a subsequent 24-hour period in which caffeine was ingested in a dosage of 1 mg/kg of body weight at intervals of one half-life during waking hours. Group 1 was composed of 18 clinically normal subjects; group 2 was 18 subjects with frequent ventricular ectopic beats (VEBs) and no (n = 16) or minor (n = 2) cardiac disease. Sinus rhythm was the prevailing rhythm in all subjects at all times. For group 1, the mean sinus rate during the caffeine-free period was 77 + or - 10 beats per minute, compared to 73 + or - 9 beats per minute during the period of caffeine ingestion (not significant). Similarly, for group 2, the average sinus rate during the caffeine-free period was 76 + or - 11 beats per minute, not significantly different from the average sinus rate during the test period, 76 + or - 10 beats per minute. During abstention from caffeine, four of 18 subjects in group 1 had infrequent (<1/hr) VEBs, compared to nine of 18 during caffeine ingestion (not significant). In group 2, some of the 16 of the 18 subjects had VEBs during the caffeine-free period, with the frequencies varying from less than one VEB per hour to 1,449 VEBs per hour. During the test period, 14 of the 18 subjects in group 2 increased their VEB frequency, and the groups's mean frequency rose from 207 + or - 350 VEBs per hour (control period) to 307 + or - 414 VEBs per hour (test period) ($p < 0.01$). The Q-T interval in group 1, measured as the corrected Q-T interval (Q-T(c), averaged 0.430 + or - 0.027 during the caffeine-free period, not significantly different from the test period (0.425 + or - 0.019). The comparable Q-T(c) values for group 2 were 0.424 + or - 0.018 during the caffeine-free period and 0.433 + or - 0.025 for the period of caffeine ingestion (not significant). Thus, these data also suggest that in moderate doses and in the absence of major underlying cardiac disease, caffeine does not significantly affect prevailing cardiac rhythm or rate, nor the mean rate of ventricular repolarization; however, the data also suggest, that caffeine can have a ventricular dysrhythmogenic effect, and although the clinical significance is still not completely certain, it seems reasonable to use it with caution in patients who may be at increased risk from ventricular dysrhythmia.

2050798068

Source: https://www.industrydocuments.ucsf.edu/docs/hlgl0000

77

AN- <DIALOG> 5565940
TI- Qualitative measurement of drugs
AU- Westerman S.T. Golz A. Komorowski F.S. Gilbert L.M.
CS- Hahnemann Medical College and Hospital, Shrewsbury, NJ
CS- USA
SO- <JN> LARYNGOSCOPE
CP- USA
SO- <PY> 1984
SO- <VO> 94/2I (165-170)
LA- ENGLISH
AB- It has been noted for several years that certain drugs produce abnormal test results in patients who are administered the electronystagmograph (ENG) test. Drug specific waveforms, termed drug evoked potentials (DEP), have been identified by using the ENG for alcohol, marijuana, cocaine, tranquilizers, amphetamines, barbiturates, opiates, hallucinogens, caffeine, aminophylline, dopamine, several gases and approximately 75 combinations of drugs (N = 821). Tests for double diagnosis of drug intake as well as tests for repeatability within the same subject were conducted. Subjects included surgical candidates, healthy volunteers, and neonates (N = 248). Drugs tested were ketamine hydrochloride, fentanyl, sodium pentothal, cocaine, dextroamphetamine, sodium seconal, morphine sulfate, phenobarbital, and meperidine. Placebos of dextrose in water and sucrose tablets were also used (N = 25). Analysis of the waveforms produced by surgical candidates and healthy volunteers revealed no false negatives or false positives. Analysis of waveforms produced by neonates whose mothers had been administered meperidine demonstrated a diminishing effect over the course of a 3 day period. Results of repeated studies revealed reliability within subject. One subject who was administered dextroamphetamine did not produce a wave consistent with amphetamine with a dosage of 5 mg. He did, however, produce an amphetamine wave when administered 10 mg of dextroamphetamine. These findings suggest a dose related effect, indicating the need for establishment of minimal levels of detection. Results indicate that with refinement and further research, this method could be used for identification of drugs diagnostically.

2050798069

Source: https://www.industrydocuments.ucsf.edu/docs/hlgl0000

78

AN- <DIALOG> 5408205
TI- The health consequences of caffeine
AU- Curatolo P.W. Robertson D.
CS- Div. Clin. Pharmacol., Dep. Med., Vanderbilt Univ. Med. Cent., Nashville, TN 37232
CS- USA
SO- <JN> ANN. INTERN. MED.
CP- USA
SO- <PY> 1983
SO- <VO> 98/5 (641-653)
LA- ENGLISH
AB- Acutely administered caffeine modestly increases blood pressure, plasma catecholamine levels, plasma renin activity, serum free fatty acid levels, urine production, and gastric acid secretion. It alters the electroencephalographic spectrum, mood, and sleep patterns of normal volunteers. Chronic caffeine consumption has no effect on blood pressure, plasma catecholamine levels, plasma renin activity, serum cholesterol concentration, blood glucose levels, or urine production. Caffeine does not appear to be useful for increasing the motility of hypomotile sperm in artificial insemination or in the therapy of minimal brain dysfunction, cancer, or Parkinson's syndrome, but it may be effective as a topical treatment of atopic dermatitis and as systemic therapy for neonatal apnea. Caffeine does not seem to be associated with myocardial infarction; lower urinary tract, renal, or pancreatic cancer; teratogenicity; or fibrocystic breast disease. The role of caffeine in the production of cardiac arrhythmias or gastric or duodenal ulcers remains uncertain.

79

AN- <DIALOG> 5241134
TI- Cortical-evoked potentials and saliva levels as basis for the comparison of pure analgesics to analgesic combinations
AU- Derendorf H. Drehsen G. Rohdewald P.
CS- Coll. Pharm., Univ. Florida, Gainesville, FL
CS- USA
SO- <JN> PHARMACOLOGY
CP- SWITZERLAND
SO- <PY> 1982
SO- <VO> 25/4 (227-236)
LA- ENGLISH
AB- Cortical-evoked potentials, pain ratings and thresholds of sensation were monitored after tooth pulp stimulation under the influence of marketed mixtures of analgesics. Simultaneously the saliva levels of the individual pharmacological active components of these mixtures were measured by high-performance thin-layer chromatography. The action of the mild analgesics in the mixtures was calculated from the linear correlations of changes in cortical-evoked potential with saliva levels of the analgesics. The ineffectiveness of low doses of salicylamide in the combinations was demonstrated by this method. None of the investigated combination products was superior to the same doses of either acetylsalicylic acid, paracetamol or phenazone.

2050298020

Source: https://www.industrydocuments.ucsf.edu/docs/hlgl0000

80

AN- <DIALOG> 5088843
TI- Sleep spindles: Pharmacological effects in humans
AU- Hirshkowitz M. Thornby J.I. Karacan I.
CS- Dept. Psychiat., Baylor Coll. Med., Texas Med. Cent., Houston, TX
CS- USA
SO- <JN> SLEEP
CP- USA
SO- <PY> 1982
SO- <VO> 5/1 (85-94)
LA- ENGLISH
AB- In the present paper we summarize the effects of a variety of drugs on sleep spindle activity in humans. The data are derived from 16 studies conducted in our laboratories during the past several years. The Sleep Analyzing Hybrid Computer was utilized to score each record for sleep stage and spindle activity. To the best of our knowledge, this collection of data is the largest currently available on the relationship between drug administration and standardized sleep spindle parameters in normal human subjects. Ten of the studies are dose-response evaluations. We consider this design a powerful and useful approach for basic sleep pharmacology research. Marked spindle enhancement was produced by flurazepam, WE-941 (a new benzodiazepine hypnotic), methaqualone, and secobarbital. Smaller differences were found for atropine, scopolamine, and caffeine. The results are discussed with respect to drug-class differences, sleep induction properties, the current literature, and the benzodiazepine hypothesis. Some of our future research plans with respect to spindle pharmacology also are presented.

81

AN- <DIALOG> 2587333
TI- Studies on sleep and waking in normal man
TI- <ORIGINAL> ETUDE DU SOMMEIL ET DE LA VIGILANCE CHEZ L'HOMME SAIN
AU- Nicholson A.N. Stone B.M.
CS- Roy. AF Inst. Aviat. Med., Farnborough
CS- UNITED KINGDOM
SO- <JN> CONCOURS MED.
CP- FRANCE
SO- <PY> 1981
SO- <VO> 103/28 Suppl. (44-48)
LA- FRENCH

2050798071

Source: https://www.industrydocuments.ucsf.edu/docs/hlgl0000

82

AN- <DIALOG> 2527840
TI- The use of electrophysiological techniques to project typical psychotropic drug effects: Some examples
AU- Herrmann W.M. Kubicki St.
CS- Fachgebiet Psychiat. Neurol., Abt. Exp. Klin. Pharmakol., Inst. Arzneimittel Bundesgesundh. Amt, 100 Berlin 30
CS- GERMANY, WEST
SO- <JN> EEG EMG Z. ELEKTROENZEPHALOGR. VERW. GEB.
CP- GERMANY, WEST
SO- <PY> 1981
SO- <VO> 12/1 (21-32)
LA- GERMAN
SL- ENGLISH
AB- A long existing hypothesis, i.e. that the EEG effects, in lead Osub 2Asub 2, of typical psychotropic drugs are substance class specific, when given as single oral dosages to healthy volunteers, is discussed. Using the technique of pharmacoelectroencephalography, five typical, representative neuroleptics, anxiolytics, antidepressives and psychostimulants as well as placebo were investigated in 75 volunteers, each receiving one representative of each substance class in a double blind 5 phase change over latin square design. A five minute EEG record for lead Osub 2Asub 2 was obtained pre, 1 hr and 3 hr post drug intake under RR (Relaxed Recording) conditions. Parametrisation was done using power spectrum analysis. Then 3 x 7 target variables were formed - six relative power values in predetermined frequency bands and the total power between 1.5 and 30.0 Hz on 3 occasions of measurement. Using a (five-group) linear discrimination analysis the substance effects on the EEG were transformed into 5 probability measures for the five substance classes. Some typical examples, using single subjects, of the projection of substance effects onto an electrophysiological level using a vector of 21 components (7 target variables for 3 occasions of measurement) as are presented in a table. The transformation into probability measures of the five substance classes are also shown. Five examples of projections of substance effects, which do not fit into the classifications to which they belong are presented separately. An attempt is made to explain whether single target variables from the power spectrum can contribute differently to the discrimination between single substances of different substance classes. Within the accepted system of 4 psychotropic drug classes, the following variables seem to be of importance: benzodiazepine anxiolytics show a marked increase in betasub 1(F) (12.0-18.0 Hz) power and activity and, related to sedation, an increase in delta(F) power and a decrease in alpha-power. Psychostimulants of the amphetamine type show an increase in total power and alpha-power, an increase in the power of the frequency ranges near to the alpha-band (slow beta, fast theta) and a decrease in delta power is high in the pre-values, indicating a stabilization in vigilance. The neuroleptics show a marked increase in betasub 1(F) (5.5-8.5 Hz) power, some increase in the delta(F) and a decrease in the betasub 1(F) and betasub 3(F) power; tricyclic antidepressants show an interaction between delta(F) (F) alpha - and beta-power, in the sense of a dissociative shift in vigilance.

2050798072

Source: https://www.industrydocuments.ucsf.edu/docs/hlgl0000

83

AN- <DIALOG> 2307847
TI- Quantification of the attenuation of pain sensation through evoked potentials after the application of mild analgesics
TI- <ORIGINAL> QUANTIFIZIERUNG DER DAMPFUNG IN DER SCHMERZEMPFINDUNG MIT HILFE EVOZIERTER POTENTIALE NACH GABE SCHWACHER ANALGETIKA
AU- Rohdewald P. Derendorf H. Elger C.E. Knoll O.
CS- Inst. Pharmazeut. Chem., Westfal. Wilhelms-Univ., 4400 Munster
CS- GERMANY, WEST
SO- <JN> EEG EMG Z. ELEKTROENZEPHALOGR. VERW. GEB.
CP- GERMANY, WEST
SO- <PY> 1980
SO- <VO> 11/4 (199-204)
LA- GERMAN
SL- ENGLISH
AB- Evoked potentials resulting from stimulation of tooth pulp with rectangular impulses were chosen for the indication of the analgesic activity of mild analgesics. If the stimuli were perceived subjectively as equally painful, the amplitudes of the evoked potentials were intraindividually well reproducible. The results are reproducible within one day as well as on different days. According to the large interindividual variation in the amplitude of the evoked potentials, the percentile reduction of the amplitude was a better measure for the analgesic action than the absolute height of the signal. Interindividually, the latencies have shown clear differences whereas they were reproducible for the individual. The curves for: the percentile reduction of the evoked potentials, the dimunition of the subjective pain response, and the increase in the sensitivity threshold after the application of the drug were principally similar. The results indicate, that the evoked potentials may represent a more sensitive measure of experimentally induced pain than subjective methods.

2050298023

Source: https://www.industrydocuments.ucsf.edu/docs/hlgl0000

84

AN- <DIALOG> 1428197
TI- Effect of flurazepam, pentobarbital, and caffeine on arousal threshold
AU- Bonnet M.H. Webb W.B. Barnard G.
CS- Univ. Florida, Gainesville, Fla. 32611
CS- USA
SO- <JN> SLEEP
CP- USA
SO- <PY> 1979
SO- <VO> 1/3 (271-279)
LA- ENGLISH
AB- After laboratory and procedure adaptation, 6 normal subjects were randomly administered 30 mg flurazepam (twice), 100 mg pentobarbital (twice), 400 mg caffeine (once), and placebo (twice) on non-consecutive nights. On each night subjects were aroused from standard segments of stage 2 sleep five to eight times with an ascending series of 1,000 Hz tones produced by an audiometer. Arousal threshold and awake threshold after each arousal were measured. Both thresholds were increased by flurazepam and pentobarbital and decreased by caffeine. All of the drugs appeared to modify arousal threshold in a time course fashion such that extreme effects were found during the first half of the night. However, the modifications of waking threshold by caffeine and flurazepam continued throughout the sleep period. The method may be a means of measuring the behavioral time course of drug activity during the sleep period.

85

AN- <DIALOG> 1323687
TI- Development of a classification rule for four clinical therapeutic psychotropic drug classes with EEG power-spectrum variables of human volunteers
AU- Herrmann W.M. Fichte K. Itil T.M. Kubicki St.
CS- Dept. Clin. Neuropsychopharmacol., Schering AG, Berlin/Bergkamen
CS- GERMANY, WEST
SO- <JN> PHARMAKOPSYCHIATRIE / NEURO-PSYCHOPHARMAKOLOGIE
CP- GERMANY, WEST
SO- <PY> 1979
SO- <VO> 12/1 (20-34)
LA- ENGLISH
SL- GERMAN
AB- An objective rule for the classification of psychotropic substances has been developed. Classification is based on data from 5 basic studies simultaneously designed and performed and involving 75 healthy volunteers who ingested 20 different psychotropic drugs and 5 placebos in single oral dosages. Each volunteer took one psychostimulant, one antidepressant, one neuroleptic, one minor tranquilizer and one placebo in a double-blind Latin square cross-over design. The variables were 6 frequency bands, based on power spectrum estimates and determined by factor analysis, plus total power in the 1.5-30.0 Hz range. An objective classification rule was established by multi-group (5 groups) linear discriminant analysis. Reclassification of the substances by the new rule yielded correct results for 17 out of 20 psychotropic drugs and 4 out of 5 placebos. Of placebos from various studies not used for the establishment of the classification rule, 7 of 9 were classified correctly. The validity of the rule for other classes of substances will have to be verified in independent studies.

2050798074

Source: https://www.industrydocuments.ucsf.edu/docs/hlgl0000

86

AN- <DIALOG> 1014269
TI- Human pharmacology of antidepressives
AU- Lader M.
CS- Dept. Psychiat., Inst. Psychiat., London
CS- UNITED KINGDOM
SO- <JN> BR. J. CLIN. PHARMACOL.
CP- ENGLAND
SO- <PY> 1977
SO- <VO> 4/suppl. 2 (135S-141S)
LA- ENGLISH
AB- As both the tricyclic antidepressives and the monoamine oxidase (MAO) inhibitors were discovered accidentally, their pharmacology has been established on an empirical post hoc basis only. There are no convincing models of depression in the normal human. The classification of depression remains controversial; also the relationship between clinical depression, depressive reactions and pessimistic personality traits is unresolved. Antidepressive effects are demonstrable in lugubrious normals. Pharmacokinetic and drug interaction data are important in the usage of a drug and are available from studies in normals. Secondary psychotropic effects such as sedation can be readily studied both in normals and in patients. The antidepressives produce characteristic changes in the electroencephalogram (EEG). Unwanted effects are often related to autonomic actions which can be readily quantified, salivation being most extensively studied. Psychological impairment is dose related in normals but lessens over a course of treatment in patients. Biochemical measures have been little explored despite their obvious relevance. MAO activity in platelets is diminished by MAO inhibitors. Tricyclic drugs inhibit the uptake of amines into neurones and platelets, and this inhibition can be assessed in various ways. Drug experiments in normal subjects provide data which complement, amplify and elucidate observations in patients without ever substituting for them.

Source: https://www.industrydocuments.ucsf.edu/docs/hlgl0000

87

AN- <DIALOG> 855462
TI- Dose related sleep disturbances induced by coffee and caffeine
AU- Karacan I. Thornby J.I. Anch A.M. et al.
CS- Dept. Psychiat., Sleep Disorders Cent., Baylor Coll. Med., Houston, Tex. 77030
CS- USA
SO- <JN> CLIN.PHARMACOL.THER.
CP- USA
SO- <PY> 1976
SO- <VO> 20/6 (682-689)
LA- ENGLISH
AB- In a 13 night sleep laboratory study, each of 18 normal young adult males twice received 1 cup of warm water, 1, 2, and 4 cup equivalents of regular coffee, a 4 cup equivalent of decaffeinated coffee, and a 4 cup equivalent of caffeine. All beverages were administered 30 min before bedtime according to a balanced Latin square design. Regular coffee produced dose related changes in most standard electroencephalogram electrooculogram (EEG EOG) sleep parameters, and the 4 cup equivalents of regular coffee and caffeine produced equivalent effects. Decaffeinated coffee had no effect. Regular coffee and caffeine caused rapid eye movement (REM) sleep to shift to the early part of the night and stages 3 and 4 sleep to shift to the later part. Coffee also produced dose related changes in several subjective estimates of sleep characteristics. These results suggest that coffee and caffeine may be used in normal subjects to induce symptoms mimicking those of insomnia. Such a tool should promote further understanding of insomnia.

Source: https://www.industrydocuments.ucsf.edu/docs/hlgl0000

FROM MEDLINE {DIALOG: FILE 155}

88

AN- <DIALOG> 06367760
TI- Topographic brain mapping of EEG in neuropsychopharmacology--Part II. Clinical applications (pharmaco EEG imaging).
AU- Saletu B Anderer P Kinsperger K Grunberger J
SO- <JN> Methods Find Exp Clin Pharmacol
CP- SPAIN
SO- <PY> Jun 1987
SO- <VO> 9 (6) p385-408
SN- 0379-0355
LA- ENGLISH
DT- JOURNAL ARTICLE
AB- Multi-lead analysis of drug-induced changes in human brain function as monitored by the scalp-recorded electroencephalogram (EEG) and subsequent imaging of topographic pharmaco-EEG maps has become possible and practical with the advent of modern mini- and micro-computers. The present paper gives a survey about topographic analyses of pharmaco-EEG data obtained in systematic double-blind, placebo-controlled studies in normal healthy volunteers involving representative drugs of 5 different psychopharmacological classes, such as neuroleptics (chlorprothixene), antidepressants (imipramine), tranquilizers (diazepam), psychostimulants (caffeine), and antihypoxidotics/nootropics (pyritinol). The determination of cerebral bioavailability utilizing time- and dose-efficacy relations, as well as the evaluation of bioequipotency of different formulations of compounds is shown. Topographic pharmaco-EEG seems not only to confirm our previous knowledge that quantitative analysis of the human EEG in combination with certain statistical procedures ("quantitative pharmaco-EEG") is a valuable method to determine if, how, when and at which dosage a drug will be centrally effective, but also to have the potential to show effects of psychotropic drugs on the target organ--the human brain--which could not be previously picked up by single lead recordings. Topographic pharmaco-EEG imaging can be viewed as an enlargement of our armamentarium to better understand the mode of action of psychotropic drugs in human neuropsychopharmacology, as well as to monitor and (eventually) predict therapeutic efficacy in patients.

89

AN- <DIALOG> 00847124
TI- [Dynamics of the reactivity and adaptation in the visual and motor analyzers of the cerebral cortex in normal subjects and in catatonic schizophrenia with a prolonged unfavorable course]
TI- <Original> Dinamika reaktivnosti i adaptatsii v zritel'nom i dvigatel'nom analizatorakh kory golovnogo mozga v norme i pri katationicheskoi forme shizofrenii s neblagopriiatnym zatiazhym techeniem.
AU- Gushanskii EL
SO- <JN> Zh Nevropatol Psikhiatr
CP- USSR
SO- <PY> 1966
SO- <VO> 66 (2) p242-8
LA- RUSSIAN
DT- JOURNAL ARTICLE

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Source: https://www.industrydocuments.ucsf.edu/docs/hlgl0000

FROM **SCISEARCH** {ISI's *Science Citation Index*; DIALOG: File 434}

90

GA- HT693
NR- 38
TI- THE EFFECT OF NEUROLEPTICS ON COGNITIVE AND PSYCHOMOTOR FUNCTION - A PRELIMINARY-STUDY IN HEALTHY-VOLUNTEERS
AU- KING DJ HENRY G
CS- QUEENS UNIV BELFAST,DEPT THERAPEUT & PHARMACOL,WHITLA MED BLDG,97 LISBURN RD/BELFAST BT9 7BL/ANTRIM/NORTH IRELAND/ HOLYWELL HOSP/ANTRIM//NORTH IRELAND/
SO- <JN> BRITISH JOURNAL OF PSYCHIATRY
SO- <PY> 1992
SO- <VO> 160
SO- <IS> MAY
SO- <IS> MAY
SO- <PG> 647-653
LA- ENGLISH
DT- ARTICLE
AB- The effects of haloperidol (1 mg), benzhexol (5 mg), diazepam (10 mg) and caffeine (400 mg) on subjective and objective measures of cognitive and psychomotor function were compared with placebo in 20 healthy volunteers. While both diazepam and benzhexol were associated with highly significant impairments in subjective alertness, critical flicker fusion threshold and choice reaction time (CRT), haloperidol could not be distinguished from placebo in most tests but was actually associated with an apparent improvement in CRT (in males) and simple visual reaction time. The perceptual maze test detected impairment by benzhexol on processing speed but was not sensitive to any other drug effects. Multiple-dose studies are required to establish if there is a true activating effect of haloperidol using a test of sustained attention. No effect of Eysenck personality subtype or life events on baseline or drug response data was detected.

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Source: https://www.industrydocuments.ucsf.edu/docs/hlgl0000

91

GA- FU571
NR- 210
TI- ASSESSMENT OF ABUSE LIABILITY OF STIMULANT-DRUGS IN HUMANS - A METHODOLOGICAL SURVEY
AU- FOLTIN RW FISCHMAN MW
CS- JOHNS HOPKINS UNIV,SCH MED,DEPT PSYCHIAT & BEHAV SCI,DIV BEHAV BIOL,600 N WOLFE ST,HOUCK E-2/BALTIMORE//MD/21205
SO- <JN> DRUG AND ALCOHOL DEPENDENCE
SO- <PY> 1991
SO- <VO> 28
SO- <IS> N1
SO- <PG> 3-48
LA- ENGLISH
DT- REVIEW
AB- Stimulant drugs have been illicitly consumed for non-medical purposes for centuries. Within the past several decades procedures for assessing the abuse liability of a compound prior to marketing have been developed, which should limit the introduction of new stimulants with high abuse liability to the public. Drugs with high abuse liability are positive reinforcers in that they maintain behavior leading to their consumption. Assessment of abuse liability has traditionally involved a comparison of the profile of effects of a known drug of abuse to that of a drug with unknown abuse liability. With respect to stimulant drugs, amphetamine and cocaine may be considered prototypic stimulants with high abuse liability. Various procedures have been used to study abuse liability including the measuring of (1) subjective effects, (2) drug 'liking', (3) estimates of value, (4) the accuracy of drug identification, (5) the ability of trained subjects to discriminate one drug from another and (6) drug self-administration. In this paper the behavioral effects of a number of stimulant drugs will be compared along these dimensions and the effects along each dimension will be related to each drugs' known abuse liability. Dissociations among the various measures will also be described, and the implications of these dissociations discussed. We conclude that the best single assessment of abuse liability is obtained from drug self-administration studies, but that the most accurate assessment of abuse liability is obtained when the effects of a drug are evaluated along as many of these dimensions as possible.

Source: https://www.industrydocuments.ucsf.edu/docs/hlgl0000

CAFFEINE & EEG/EP/ERP ETC REVIEWS, BOOK CHAPTERS, MONOGRAPHS, ETC
FROM MEDLINE {DIALOG: FILE 155}

92

AN- <DIALOG> 08296556
TI- Caffeine and the central nervous system: mechanisms of action, biochemical, metabolic and psychostimulant effects.
AU- Nehlig A Daval JL Debry G
CS- INSERM U 272 Universite de Nancy I, France.
SO- <JN> Brain Res Brain Res Rev
CP- NETHERLANDS
SO- <PY> May-Aug 1992
SO- <VO> 17 (2) p139-70
SN- 0165-0173
LA- ENGLISH
DT- JOURNAL ARTICLE REVIEW REVIEW, TUTORIAL
AB- Caffeine is the most widely consumed central-nervous-system stimulant. Three main mechanisms of action of caffeine on the central nervous system have been described. Mobilization of intracellular calcium and inhibition of specific phosphodiesterases only occur at high non-physiological concentrations of caffeine. The only likely mechanism of action of the methylxanthine is the antagonism at the level of adenosine receptors. Caffeine increases energy metabolism throughout the brain but decreases at the same time cerebral blood flow, inducing a relative brain hypoperfusion. Caffeine activates noradrenaline neurons and seems to affect the local release of dopamine. Many of the alerting effects of caffeine may be related to the action of the methylxanthine on serotonin neurons. The methylxanthine induces dose-response increases in locomotor activity in animals. Its psychostimulant action on man is, however, often subtle and not very easy to detect. The effects of caffeine on learning, memory, performance and coordination are rather related to the methylxanthine action on arousal, vigilance and fatigue. Caffeine exerts obvious effects on anxiety and sleep which vary according to individual sensitivity to the methylxanthine. However, children in general do not appear more sensitive to methylxanthine effects than adults. The central nervous system does not seem to develop a great tolerance to the effects of caffeine although dependence and withdrawal symptoms are reported.
RF- 656

2050798080

Source: https://www.industrydocuments.ucsf.edu/docs/hlgl0000

93

AN- <DIALOG> 08150823
TI- Sleep disorders in geriatric patients.
AU- Wooten V
CS- Sleep Disorders Center, St. Vincent Infirmary Medical Center, Little Rock, Arkansas.
SO- <JN> Clin Geriatr Med
CP- UNITED STATES
SO- <PY> May 1992
SO- <VO> 8 (2) p427-39
SN- 0749-0690
LA- ENGLISH
DT- JOURNAL ARTICLE REVIEW REVIEW, TUTORIAL
AB- The elderly have more organic sleep problems disturbing sleep and contributing to insomnia than younger individuals. The most common disorders afflicting the elderly are obstructive sleep apnea, restless legs syndrome, and nocturnal myoclonus. Poor sleep habits often aggravate or contribute to the ongoing difficulty with sleeping. In the depressed elderly, characteristic EEG changes occur that may help distinguish major depression from pseudodementia; however, it should be considered that pseudodementia may be a harbinger of primary dementia. A careful sleep history and often evaluation by polysomnography are central to the management of sleep problems in the elderly. In conjunction with treatment of any underlying organic sleep disorders, brief administration of short-acting benzodiazepine sedatives for sleep onset insomnia or rapid-acting intermediate half-life benzodiazepines for sleep maintenance insomnia can be quite helpful in the elderly, especially if behavioral techniques also are employed. Elimination of medications, alcohol, and caffeine, which disturb sleep, is also an important part of the treatment approach.
RF- 83

2050798081

Source: https://www.industrydocuments.ucsf.edu/docs/hlgl0000

94

AN- <DIALOG> 07765154
TI- Receptor-activated cytoplasmic Ca2+ oscillations in pancreatic acinar cells: generation and spreading of Ca2+ signals.
AU- Petersen OH Gallacher DV Wakui M Yule DI Petersen CC Toescu EC
CS- MRC Secretory Control Research Group, University of Liverpool, UK.
SO- <JN> Cell Calcium
CP- SCOTLAND
SO- <PY> Feb-Mar 1991
SO- <VO> 12 (2-3) p135-44
SN- 0143-4160
LA- ENGLISH
DT- JOURNAL ARTICLE REVIEW REVIEW, TUTORIAL
AB- Receptor-activated cytoplasmic Ca2+ oscillations have been investigated using both single cell microfluorometry and voltage-clamp recording of Ca(2+)-dependent Cl- current in single internally perfused acinar cells. In these cells there is direct experimental evidence showing that the ACh-evoked [Ca2+]i fluctuations are due to an inositol trisphosphate-induced small steady Ca2+ release which in turn evokes repetitive Ca2+ spikes via a caffeine-sensitive Ca(2+)-induced Ca2+ release process. There is indirect evidence suggesting that receptor-activation in addition to generating the Ca2+ releasing messenger, inositol trisphosphate, also produces another regulator involved in the control of Ca2+ signal spreading. Intracellular inositol trisphosphate or Ca2+ infusion produce short duration repetitive spikes confined to the cytoplasmic area close to the plasma membrane, but these signals can be made to progress throughout the cell by addition of caffeine or by receptor activation.
RF- 44

2050798082

Source: https://www.industrydocuments.ucsf.edu/docs/hlgl0000

95

AN- <DIALOG> 07557388
TI- Caffeine and cardiac arrhythmias.
AU- Myers MG
CS- Division of Cardiology, Sunnybrook Health Science Centre, Toronto, Ontario.
SO- <JN> Ann Intern Med
CP- UNITED STATES
SO- <PY> Jan 15 1991
SO- <VO> 114 (2) p147-50
SN- 0003-4819
LA- ENGLISH
DT- JOURNAL ARTICLE REVIEW REVIEW, TUTORIAL
AB- PURPOSE: To review the evidence supporting the belief that caffeine causes cardiac arrhythmias. DATA SOURCES: Studies published since 1982 identified through computerized searches of MEDLINE, TOXLINE, and Chemical Abstracts and a review of bibliographies of relevant articles on the subject of caffeine and cardiac arrhythmias. STUDY SELECTION: All clinical studies examining caffeine as a cause of cardiac arrhythmias and a selection of basic science experiments to illustrate caffeine's effects in vitro. DATA EXTRACTION: Study quality was assessed and all available clinical data pertaining to caffeine as a cause of arrhythmias were summarized. RESULTS OF DATA ANALYSIS: In one electrophysiologic study, caffeine was associated with an increased susceptibility to provoked cardiac arrhythmias. In five placebo-controlled trials, caffeine in doses up to 500 mg daily (equivalent to 5 to 6 cups of coffee) did not increase the frequency or severity of ventricular arrhythmias. One large epidemiologic study reported an increase in the frequency of ventricular extrasystoles in persons consuming 9 or more cups of coffee daily. CONCLUSION: Moderate ingestion of caffeine does not increase the frequency or severity of cardiac arrhythmias in normal persons, patients with ischemic heart disease, or those with pre-existing serious ventricular ectopy.
RF- 23

96

AN- <DIALOG> 05809898
TI- The physiological role of adenosine in the central nervous system.
AU- Dunwiddie TV
SO- <JN> Int Rev Neurobiol
CP- UNITED STATES
SO- <PY> 1985
SO- <VO> 27 p63-139
SN- 0074-7742
LA- ENGLISH
DT- JOURNAL ARTICLE REVIEW
RF- 245

2050798083

Source: https://www.industrydocuments.ucsf.edu/docs/hlgl0000

97

AN- <DIALOG> 05264526
TI- Ca2+ and the sarcoplasmic reticulum.
AU- Nayler WG Dresel PE
SO- <JN> J Mol Cell Cardiol
CP- ENGLAND
SO- <PY> Feb 1984
SO- <VO> 16 (2) p165-74
SN- 0022-2828
LA- ENGLISH
DT- JOURNAL ARTICLE REVIEW
AB- The role of the sarcoplasmic reticulum in the regulation of cytosolic calcium is briefly reviewed. A method is described for monitoring calcium efflux from relatively large amounts of SR in a newly designed filtration apparatus using membrane filtration. Two phases of Ca2+ efflux from SR preloaded in the presence of oxalate were observed: an early exponential phase, followed by a phase of constant efflux. Changes in pH from 6.8 to 7.4 resulted in increased efflux. A change in the opposite direction resulted in an initial rapid increase in efflux before the expected decrease occurred. Caffeine (10 and 20 mM) caused increased efflux. Addition of 100 microM EGTA to the buffer resulted in major changes in the exponential phase but not in the phase of constant release, and potentiated the effect of caffeine. The method appears suitable for a variety of pharmacological studies in SR as well as other tissue fractions.
RF- 60

98

AN- <DIALOG> 05221809
TI- Pharmacology of excitation-contraction coupling in skeletal muscle.
AU- Lakshminarayanaiah N Bianchi CP
SO- <JN> Rev Pure Appl Pharmacol Sci
CP- ENGLAND
SO- <PY> Jan-Mar 1983
SO- <VO> 4 (1) p27-54
SN- 0197-2839
LA- ENGLISH
DT- JOURNAL ARTICLE REVIEW
RF- 148

2050798084

Source: https://www.industrydocuments.ucsf.edu/docs/hlgl0000

99

AN- <DIALOG> 03657425
TI- Drug research and human sleep.
AU- Oswald I
SO- <JN> Prog Drug Res
CP- SWITZERLAND
SO- <PY> 1978
SO- <VO> 22 p355-72
SN- 0071-786X
LA- ENGLISH
DT- JOURNAL ARTICLE REVIEW
RF- 91

100

AN- <DIALOG> 03082130
TI- On the electrical and mechanical properties of the longitudinal and circular muscles of the guinea-pig ileum. pp. 197-214.
AU- Kuriyama H Suzuki H
SO- <JN> In: Worcel M, Vassort G, ed. Smooth muscle pharmacology and physiology. Paris, INSERM, 1976. WE 500 S668 1975.
CP- FRANCE
CA- WE 500 S668 1975
LA- ENGLISH
DT- MONOGRAPH

101

AN- <DIALOG> 02562577
TI- The electrophysiology of gastrointestinal chemoreceptors.
AU- Davison JS
SO- <JN> Digestion
CP- SWITZERLAND
SO- <PY> 1972
SO- <VO> 7 (5) p312-7
SN- 0012-2823
LA- ENGLISH
DT- JOURNAL ARTICLE REVIEW
RF- 19

2050798085

Source: https://www.industrydocuments.ucsf.edu/docs/hlgl0000

102

AN- <DIALOG> 02208006
TI- Heart: excitation-contraction coupling.
AU- Langer GA
SO- <JN> Annu Rev Physiol
CP- UNITED STATES
SO- <PY> 1973
SO- <VO> 35 p55-86
SN- 0066-4278
LA- ENGLISH
DT- JOURNAL ARTICLE REVIEW
RF- 117

103

AN- <DIALOG> 00801429
TI- Paroxysmal dystonic choreoathetosis. A family study and review of the literature.
AU- Richards RN Barnett HJ
SO- <JN> Neurology
CP- UNITED STATES
SO- <PY> May 1968
SO- <VO> 18 (5) p461-9
SN- 0028-3878
LA- ENGLISH
DT- JOURNAL ARTICLE

FROM SCISEARCH {ISI's *Science Citation Index*; DIALOG: File 434}

104

GA- NP228
NR- 178
TI- DO BENZODIAZEPINES HAVE A ROLE IN CHRONIC PAIN MANAGEMENT
AU- DELLEMIJN PLI FIELDS HL
CS- UNIV CALIF SAN FRANCISCO,DEPT NEUROL M794,BOX 0114/SAN FRANCISCO//CA/94143 UNIV CALIF SAN FRANCISCO,DEPT NEUROL M794/SAN FRANCISCO//CA/94143
SO- <JN> PAIN
SO- <PY> 1994
SO- <VO> 57
SO- <IS> N2
SO- <IS> MAY
SO- <PG> 137-152
SN- 0304-3959
LA- ENGLISH
DT- REVIEW

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Source: https://www.industrydocuments.ucsf.edu/docs/hlgl0000

105

GA- NF168
NR- 30
TI- THE PRESENT USE OF ELECTROCONVULSIVE-THERAPY
AU- WEINER RD KRYSTAL AD
CS- DUKE UNIV,MED CTR,DEPT PSYCHIAT/DURHAM//NC/27707 VET ADM MED CTR/DURHAM//NC/27705
SO- <JN> ANNUAL REVIEW OF MEDICINE
SO- <PY> 1994
SO- <VO> 45
SO- <PG> 273-281
SN- 0066-4219
LA- ENGLISH
DT- REVIEW
AB- Physicians attempting to treat certain severe mental disorders have recently shown renewed interest in electroconvulsive therapy (ECT). A number of technical innovations have made ECT safer, as well as more effective. These innovations include oxygenation, muscular relaxation, unilateral nondominant electrode placement, use of brief-pulse stimuli, titrated stimulus dosing, electroencephalographic (EEG) monitoring, determination of seizure adequacy, and pharmacologic enhancement of treatment response.

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Source: https://www.industrydocuments.ucsf.edu/docs/hlgl0000

106

GA- MZ469
NR- 134
TI- MODULATION BY ADRENALINE OF ELECTROPHYSIOLOGICAL MEMBRANE PARAMETERS AND CONTRACTILITY IN INTACT AND INTERNALLY PERFUSED SINGLE MUSCLE-FIBERS OF THE CRAYFISH
AU- ZACHAROVA D LIPSKA E HENCEK M HOCHMANNOVA J SAJTER V
CS- SLOVAK ACAD SCI,INST MOLEC PHYSIOL & GENET,VLARSKA 5/BRATISLAVA 83334//SLOVAKIA/ INST POSTGRAD MED EDUC/BRATISLAVA 83303//SLOVAKIA/
SO- <JN> GENERAL PHYSIOLOGY AND BIOPHYSICS
SO- <PY> 1993
SO- <VO> 12
SO- <IS> N6
SO- <IS> DEC
SO- <PG> 543-577
SN- 0231-5882
LA- ENGLISH
DT- REVIEW
AB- (1) The effect of catecholamines on basic membrane characteristics (including labeled ionic fluxes) and contractile parameters was followed in current clamp and voltage clamp conditions in intact muscle fibres and internally perfused muscle fibre segments respectively of the crayfish Astacus fluviatilis; i.e. in muscle fibres which spike and activate tension on calcium principle. (2) Both adrenaline and noradrenaline (6.10(-6) mol/l) facilitated twitch tension induced by graded membrane responses or strontium all-or-none spikes. No effect of isoprenaline was observed. (3) Adrenaline (6.10(-6) - 6.10(-5) mol/l) produced an inotropic effect, which appeared with a latency of 2 min and reached its maximum in 5 min. The rates of activation and relaxation of contraction were increased, whilst the latency and the threshold depolarization were decreased. The changes persisted (several tens of min) after washout of adrenaline, depending on concentration and duration of adrenaline application. (4) The resting potential and the strontium spike (Ca2+ replaced with Sr2+) were not influenced and the graded responses were facilitated by adrenaline (from 36.4 +/- 1 mV to 40.0 +/- 2 mV; RP;= 77.2 +/- 0.5 mV). (5) Extracellular Ca2+ ions are required for the inotropic effect of adrenaline to occur. The decrease of electrical and contractile responses in nominal calcium-free solutions or after a blockade of Ca2+ influx by Ni2+, ions (1 mmol/l) was relieved by adrenaline. The persistence of inotropic effect of adrenaline was absent, when the extracellular concentration of Ca2+ ions, [Ca2+](0) was decreased from 13.5 to 3.4 mmol/l or the Ni2+ ions were added. The influx Sr-89(2+) ions was decreased in the presence of Ni2+ ions from 24.2 +/- 4.7 pmol.cm(-2).s(-1) to 11.0 +/- 2.8 pmol.cm(-2).s(-1) but restored to 20.4 +/- 5.8 pmol.cm(-2).s(-1) in the presence of adrenaline (6 mu mol/l). (6) Adrenaline itself decreased the influx, of Sr-89(2+) ions, and prolonged the time constant of efflux both in resting and stimulated fibres. (7) The effect of adrenaline is dependent on mobilization of Ca2+ ions from the sarcoplasmic reticulum. First, the inotropic effect of adrenaline was absent in the presence of procaine (blockator of the Ca release channel of the SR), in spite of the increase of the active membrane response (all-or-none procaine action potential); second, adrenaline accelerated the uptake of Ca ions by SR as evidenced by shortening of the restitution processes after caffeine contractures by adrenaline. (8) Membrane calcium currents are increased by adrenaline as a rule; mainly at lower depolarizations (-50 to -20 mV). All components of the whole calcium conductance, which differ by time constants of activation tau(M), and inactivation tau(h) (i.e. of the fast, the intermediate and the slow calcium conductance respectively) are increased in the presence of adrenaline. The calcium conductance of the fast component was increased by 31 +/- 6.3%, of the slow component by 132.2

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Source: https://www.industrydocuments.ucsf.edu/docs/hlgl0000

+/- 27.6% and of the intermediate component by 101.5 +/- 20.9% (n = 22, P = 0.01-0.001). The enhancement of calcium currents persisted after the withdrawal of adrenaline from the bathing solution. The time constants of activation, tau(M), were not significantly changed; the time constant of inactivation, tau(h) in the fast calcium channel was prolonged.

107

GA- MV488
NR- 78
TI- ELECTROCONVULSIVE-THERAPY OF ACUTE MANIC EPISODES - A REVIEW OF 50 YEARS EXPERIENCE
AU- MUKHERJEE S SACKEIM HA SCHNUR DB
CS- MED COLL GEORGIA,DEPT PSYCHIAT,1515 POPE AVE/AUGUSTA//GA/30912 VET AFFAIRS MED CTR/AUGUSTA//GA/00000 NEW YORK STATE PSYCHIAT INST & HOSP,DEPT BIOL PSYCHIAT/NEW YORK//NY/00000 COLUMBIA UNIV COLL PHYS & SURG,DEPT PSYCHIAT/NEW YORK//NY/10032 ELMHURST HOSP CTR,DEPT PSYCHIAT/NEW YORK//NY/00000
SO- <JN> AMERICAN JOURNAL OF PSYCHIATRY
SO- <PY> 1994
SO- <VO> 151
SO- <IS> N2
SO- <IS> FEB
SO- <PG> 169-176
SN- 0002-953X
LA- ENGLISH
DT- REVIEW
AB- Objective: The most common indication for electroconvulsive therapy (ECT) is major depression. It is less recognized that ECT is effective also in the treatment of acute mania. This article aims to Provide a comprehensive and critical review of the literature on the use of ECT for manic patients. Method: All published papers in the English language on the use of ECT in acute mania that could be found were reviewed with regard to efficacy, frequency and number of treatments, bilateral versus unilateral electrode placement, predictors of antimanic response, stability of therapeutic response, cognitive consequences, and other relevant issues. Results: The evidence indicates that ECT is associated with remission or marked clinical improvement in 80% of manic patients and that it is an effective treatment for patients whose manic episodes have responded poorly to pharmacotherapy. Manic patients do not require a high frequency or prolonged course of treatments to respond to ECT. The seizure threshold appears to be lower in manic patients than in depressed patients. The issues of relapse following response to ECT, cognitive consequences of ECT, and the relative merits of unilateral versus bilateral ECT in manic patients require further study. Conclusions: ECT is an effective and safe treatment for acute mania. Remission of mania following ECT reflects a primary therapeutic effect rather than a secondary consequence of an ECT-induced organic brain syndrome.

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Source: https://www.industrydocuments.ucsf.edu/docs/hlgl0000

108

GA- MM108
NR- 1402
TI- CLINICAL USES OF BENZODIAZEPINES
AU- HOLLISTER LE MULLEROERLINGHAUSEN B RICKELS K SHADER RI
CS- UNIV TEXAS,HLTH SCI CTR,HARRIS CTY PSYCHIAT CTR,DEPT PSYCHIAT,2800 S MACGREGOR WAY/HOUSTON//TX/77225 UNIV TEXAS,HLTH SCI CTR,HARRIS CTY PSYCHIAT CTR,DEPT PHARMACOL/HOUSTON//TX/77225 FREE UNIV BERLIN,DEPT PSYCHIAT/W-1000 BERLIN//GERMANY/ UNIV PENN,DEPT PSYCHIAT/PHILADELPHIA//PA/19104 TUFTS UNIV,SCH MED,DEPT PHARMACOL & EXPTL THERAPEUT/BOSTON//MA/00000
SO- <JN> JOURNAL OF CLINICAL PSYCHOPHARMACOLOGY
SO- <PY> 1993
SO- <VO> 13
SO- <IS> N6
SO- <IS> DEC
SO- <PG> S1-S169
SN- 0271-0749
LA- ENGLISH
DT- REVIEW

109

GA- KU968
NR- 112
TI- AIRWAY MANAGEMENT AND SEDATION FOR PEDIATRIC TRANSPORT
AU- MCDONALD TB BERKOWITZ RA
CS- UNIV ILLINOIS,COLL MED,DEPT ANESTHESIOL,1740 W TAYLOR ST/CHICAGO//IL/60612 UNIV ILLINOIS,EYE & EAR INFIRM/CHICAGO//IL/60612
SO- <JN> PEDIATRIC CLINICS OF NORTH AMERICA
SO- <PY> 1993
SO- <VO> 40
SO- <IS> N2
SO- <IS> APR
SO- <PG> 381-406
SN- 0031-3955
LA- ENGLISH
DT- REVIEW

2050798090

Source: https://www.industrydocuments.ucsf.edu/docs/hlgl0000

110

GA- FL258
NR- 102
TI- CARDIAC ELECTROPHYSIOLOGY OF ADENOSINE - BASIC AND CLINICAL CONCEPTS
AU- LERMAN BB BELARDINELLI L
CS- CORNELL UNIV,MED CTR,NEW YORK HOSP,DEPT CARDIOL,525 E 68TH ST,STARR 4/NEW YORK//NY/10021 UNIV FLORIDA,COLL MED,DEPT MED,DIV CARDIOL/GAINESVILLE//FL/32611
SO- <JN> CIRCULATION
SO- <PY> 1991
SO- <VO> 83
SO- <IS> N5
SO- <PG> 1499-1509
LA- ENGLISH
DT- REVIEW

111

GA- F0732
NR- 218
TI- MINIMAL RESIDUAL NEOPLASTIC DISEASE - CONCEPT, PATHOGENESIS, AND SUPPLEMENTARY THERAPEUTIC POSSIBILITIES
AU- PRINDULL G
CS- UNIV GOTTINGEN,DEPT PEDIAT,HUMBOLDTALEE 38/D-3400 GOTTINGEN//FED REP GER/
SO- <JN> CANCER TREATMENT REVIEWS
SO- <PY> 1986
SO- <VO> 13
SO- <IS> N3
SO- <PG> 177-194
LA- ENGLISH
DT- REVIEW, BIBLIOGRAPHY

2050798091

Source: https://www.industrydocuments.ucsf.edu/docs/hlgl0000

112

GA- TH794
NR- 160
TI- THE SURPRISING HEART - A REVIEW OF RECENT PROGRESS IN CARDIAC ELECTROPHYSIOLOGY
AU- NOBLE D
CS- UNIV OXFORD,PHYSIOL LAB/OXFORD OX1 3PT//ENGLAND/
SO- <JN> JOURNAL OF PHYSIOLOGY-LONDON
SO- <PY> 1984
SO- <VO> 353
SO- <IS> AUG
SO- <PG> 1-50
LA- ENGLISH
DT- REVIEW, BIBLIOGRAPHY

113

GA- TC392
NR- 94
TI- ELECTROPHYSIOLOGIC EFFECTS OF ADENOSINE-TRIPHOSPHATE AND ADENOSINE ON THE MAMMALIAN HEART - CLINICAL AND EXPERIMENTAL ASPECTS
AU- BELHASSEN B PELLEG A
CS- HAHNEMANN UNIV,LIKOFF CARDIOVASC INST,CARDIAC ELECTROPHYSIOL LAB/PHILADELPHIA//PA/19102 ICHILOV HOSP,TEL AVIV MED CTR,CARDIAC ELECTROPHYSIOL LABS/IL-64239 TEL AVIV//ISRAEL/ LANKENAU MED RES CTR,DIV CARDIOL/PHILADELPHIA//PA/00000
SO- <JN> JOURNAL OF THE AMERICAN COLLEGE OF CARDIOLOGY
SO- <PY> 1984
SO- <VO> 4
SO- <IS> N2
SO- <PG> 414-424
LA- ENGLISH
DT- REVIEW, BIBLIOGRAPHY

2050798092

Source: https://www.industrydocuments.ucsf.edu/docs/hlgl0000

114

GA- SR783
NR- 76
TI- MECHANICAL-PROPERTIES OF CELLULAR CARDIAC PREPARATIONS
AU- DECLERCK NM BRUTSAERT DL
CS- UNIV ANTWERP,DEPT PHYSIOL & MED/ANTWERP//BELGIUM/
SO- <JN> PHARMACOLOGY & THERAPEUTICS
SO- <PY> 1984
SO- <VO> 24
SO- <IS> N1
SO- <PG> 133-146
LA- ENGLISH
DT- REVIEW, BIBLIOGRAPHY

115

GA- SP419
NR- 264
TI- CEREBRAL-CIRCULATION AND METABOLISM
AU- SIESJO BK
CS- UNIV LUND,EXPTL BRAIN RES LAB/S-22101 LUND//SWEDEN/
SO- <JN> JOURNAL OF NEUROSURGERY
SO- <PY> 1984
SO- <VO> 60
SO- <IS> N5
SO- <PG> 883-908
LA- ENGLISH
DT- REVIEW, BIBLIOGRAPHY

116

GA- RX181
NR- 234
TI- EVOKED-RESPONSES IN NORMAL AND DISEASED MUSCLE WITH PARTICULAR REFERENCE TO TWITCH POTENTIATION
AU- KRARUP C
CS- NINCDS,BLDG 10,ROOM 5N230/BETHESDA//MD/20205
SO- <JN> ACTA NEUROLOGICA SCANDINAVICA
SO- <PY> 1983
SO- <VO> 68
SO- <IS> N5
SO- <PG> 269-315
LA- ENGLISH
DT- REVIEW, BIBLIOGRAPHY

2050798093

Source: https://www.industrydocuments.ucsf.edu/docs/hlgl0000

117

GA- PZ022
NR- 123
TI- PHYSIOLOGY OF CALCIUM CURRENT IN CARDIAC-MUSCLE
AU- KEUNG ECH ARONSON RS
CS- YESHIVA UNIV ALBERT EINSTEIN COLL MED,DEPT MED,DIV CARDIOL/BRONX//NY/10461
SO- <JN> PROGRESS IN CARDIOVASCULAR DISEASES
SO- <PY> 1983
SO- <VO> 25
SO- <IS> N4
SO- <PG> 279-296
LA- ENGLISH
DT- REVIEW, BIBLIOGRAPHY

2050798094

Source: https://www.industrydocuments.ucsf.edu/docs/hlgl0000

THEOPHYLLINE, EEG/EP/ERP ETC & HEALTHY/NORMAL VOLS/SUBJECTS
FROM psycINFO {DIALOG: FILE 11}

118

AN- 00851144
TI- Effects of single and repeated doses of theophylline on aspects of performance, electrophysiology and subjective assessments in healthy human subjects.
AU- Bartel, Peter Delport, Rhena Lotz, Barend Ubbink, Johan et al
CS- HF Verwoerd Hosp, Neuropsychopharmacology Lab, Pretoria, South Africa
SO- <JN> Psychopharmacology
SO- 1992 Jan Vol 106(1) 90-96
SN- 00333158
LA- English
DT- JOURNAL ARTICLE
AB- Evaluated the effects of both single (400 mg) and repeated doses (300 mg for 4 wks) of theophylline on a battery of 9 performance tests, the EEG, the electromyogram (EMG), and on subjective assessments of mood and side effects in 20 healthy adults (aged 20-36 yrs). An information processing test revealed enhanced performance in both phases, while an addition test showed improved performance in the single dose phase only. The remaining 7 performance tests failed to show significant differences between theophylline and placebo. Single doses of theophylline did not significantly alter mood, but marked adverse effects were encountered in the repeated dose phase, possibly related to unpleasant side effects. Findings demonstrate central nervous system (CNS) stimulation by both single and repeated doses of theophylline with the occurrence of adverse side effects during repeated administrations.
AB- (PsycINFO Database Copyright 1992 American Psychological Assn, all rights reserved)

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Source: https://www.industrydocuments.ucsf.edu/docs/hlgl0000

FROM EMBASE {*Excerpta Medica*; DIALOG: File 73}

119

AN- <DIALOG> 7577260
TI- Electrophysiological indices of central and peripheral nervous system function during theophylline therapy
AU- Bartel P. Lotz B. Delport R. Ubbink J. Becker P.
CS- Department of Neurology, University of Pretoria, Pretoria
CS- South Africa
SO- <JN> NEUROPSYCHOBIOLOGY
CP- Switzerland
SO- <PY> 1989
SO- <VO> 21/2 (104-108)
SN- 0302-282X
LA- English
AB- Electrophysiological parameters including the EEG, somatosensory evoked potentials (SEP), F waves, long loop reflexes and peripheral nerve conduction velocities were assessed during the 15th week of theophylline therapy, with serum levels maintained in the low therapeutic range, in young, healthy male volunteers (n = 7). Recordings were repeated 1 week after vitamin B6 supplementation and finally 6-7 weeks after the cessation of medication. Alpha amplitudes were significantly lower during theophylline therapy compared to the posttreatment baseline recording. The SEP findings failed to reveal any significant differences between the three recordings, as was the case with peripheral nerve conduction velocities and long loop reflexes. The average F wave latencies during theophylline therapy were significantly shorter, and the percentage F waves recorded was higher, compared to the baseline recordings. These findings suggest that relatively long-term theophylline therapy has stimulatory effects on aspects of electrical activity in the brain and spinal cord. These effects appeared to be unchanged after vitamin B6 supplementation for 1 week.

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Source: https://www.industrydocuments.ucsf.edu/docs/hlgl0000

120

AN- <DIALOG> 7140022
TI- Rectum has abnormal ion transport but normal cAMP-binding proteins in cystic fibrosis
AU- Goldstein J.L. Nash N.T. Al-Bazzaz F. Layden T.J. Rao M.C.
CS- Department of Medicine, University of Illinois College of Medicine at Chicago, Chicago, IL 60612
CS- USA
SO- <JN> AM. J. PHYSIOL., CELL PHYSIOL.
CP- USA
SO- <PY> 1988
SO- <VO> 254/5 (23/5) (C719-C724)
SN- 0002-9513
LA- English
AB- The luminal membranes of involved tissues in cystic fibrosis (CF) are relatively impermeable to Cl and the regulation of Cl transport by adenosine 3',5'-cyclic monophosphate (cAMP)-mediated hormones is abnormal. We investigated the human rectum as a putative model for CF. We compared in vivo transrectal potential difference (PD) in CF and in normal subjects in response to sequential perfusions with various test solutions. The base-line PD was different in normal (-35.5 plus or minus 4.0 mV; lumen negative; mean plus or minus SE; n = 9) and CF subjects (-23.4 plus or minus 3.1 mV; n = 6; $P < 0.025$) and was eliminated by amiloride (10-4 M) perfusion in both groups by 3 min. However, in response to a Cl-free solution with amiloride, all six CF subjects exhibit less of a change in PD (PD, -2.2 plus or minus 1.2 mV vs. -11.7 plus or minus 1.5 mV in 6 controls; $P < 0.01$). Furthermore, normal subjects (n = 7) respond to a 5 mM theophylline + amiloride perfusion with an increase in lumen negative PD, whereas CF subjects (n = 6) show no increase in lumen-negative PD. Rectal biopsy specimens from four normal and four CF subjects exhibit similar (2- to 3-fold) increases in theophylline-induced cAMP content and have similar cAMP-binding proteins (CF, n = 3; control, n = 3). We conclude that the rectum is an involved epithelium in CF in which the aberration may lie at a point beyond the binding of cAMP to its protein kinase.

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Source: https://www.industrydocuments.ucsf.edu/docs/hlgl0000

121

AN- <DIALOG> 6029024
TI- Breathing during sleep
AU- Flenley D.C.
CS- Department of Respiratory Medicine, University of Edinburgh, City Hospital, Edinburgh EH10 5SB
CS- UNITED KINGDOM
SO- <JN> ANN. ACAD. MED. SINGAPORE
CP- SINGAPORE
SO- <PY> 1985
SO- <VO> 14/3 (479-484)
LA- ENGLISH
AB- Hypoxemia during the rapid eye movement phase of sleep is common in 1) older healthy normal subjects over 55 years of age; 2) the sleep apnoea syndromes - such as obstructive sleep apnoea, where oro-nasal airflow ceases for more than 10 seconds on many separate occasions throughout the night, due to failure of contraction of the genio-glossus muscle; 3) 'blue and bloated' patients with chronic bronchitis and emphysema, where profound nocturnal hypoxemia is common in REM sleep, and is associated with further elevation of pulmonary arterial pressure; 4) the overlap syndrome - where 'blue and bloated' chronic bronchitis is associated with an obstructive sleep apnoea syndrome; and 5) bronchial asthma, where hypoxemia is associated with irregular breathing and possibly nocturnal bronchoconstriction. Although absolute recognition depends upon all night sleep studies, monitoring of ear oxygen saturation, breathing patterns, and EEG, the clinical features when awake can lead to suspicion of sleep hypoxemia - as, for example, obesity and obstructive sleep apnoea with loud snoring and restlessness in sleep, hypoxemia during wakefulness in the overlap syndrome, and nocturnal awakening with wheeze in bronchial asthma. Treatment depends on the cause, and may vary from weight loss and nasal continuous positive airway pressure in obstructive sleep apnoea, to nocturnal oxygen in 'blue bloaters', a combination of these two in the overlap syndrome, and long acting bronchodilators such as slow release theophyllines in nocturnal asthma. Recognition and appropriate treatment of nocturnal hypoxemia is an important new development in respiratory medicine.

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Source: https://www.industrydocuments.ucsf.edu/docs/hlgl0000

FROM MEDLINE {DIALOG: FILE 155}

122

AN- <DIALOG> 08169288
TI- Theophylline and prostaglandin E2 on duodenal bicarbonate secretion: role for 5'-cyclic adenosine monophosphate.
AU- Mu JZ Hogan DL Koss MA Isenberg JI
CS- Department of Medicine, UCSD Medical Center, California.
SO- <JN> Gastroenterology
CP- UNITED STATES
SO- <PY> Jul 1992
SO- <VO> 103 (1) p153-9
SN- 0016-5085
LA- ENGLISH
DT- JOURNAL ARTICLE
AB- Cyclic adenosine monophosphate (cAMP) has been implicated as an intracellular "second" messenger in duodenal mucosal bicarbonate secretion in animals. The purpose of this study was to determine whether cAMP may mediate duodenal mucosal bicarbonate secretion in humans. In healthy volunteers, a 4-cm segment of proximal duodenum was isolated from gastric and pancreaticobiliary secretions. Either the phosphodiesterase inhibitor theophylline, prostaglandin (PG) E2, or a combination thereof was administered topically to the isolated duodenal mucosa. Theophylline (10(-2) mol/L) and PGE2 (10(-5)-10(-4) mol/L) each significantly increased bicarbonate secretion and transmucosal potential difference. Moreover, when theophylline and PGE2 were administered in combination, the duodenal bicarbonate output was additive compared to either agent alone. When theophylline was infused with increasing doses of PGE2, the dose-response curve was shifted to the left. Furthermore, increases in bicarbonate secretion and transmucosal potential difference were correlated significantly. These results suggest that cAMP may act as an intracellular mediator of human duodenal mucosal bicarbonate secretion.

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Source: https://www.industrydocuments.ucsf.edu/docs/hlgl0000

FROM SCISEARCH {ISI's *Science Citation Index*; DIALOG: File 434}

123

GA- FM931
NR- 26
TI- PHARMACOKINETIC-PHARMACODYNAMIC MODELING OF THE INTERACTION BETWEEN FLUMAZENIL AND MIDAZOLAM IN VOLUNTEERS BY APERIODIC EEG ANALYSIS
AU- BREIMER LTM BURM AGL DANHOF M HENNIS PJ VLETTER AA DEVOOGT JWH SPIERDIJK J BOVILL JG
CS- LEIDEN UNIV HOSP,DEPT ANAESTHESIOL,POB 9600/2300 RC LEIDEN//NETHERLANDS/ LEIDEN STATE UNIV,CTR BIOPHARMACEUT SCI,DIV PHARMACOL/2312 AV LEIDEN//NETHERLANDS/
SO- <JN> CLINICAL PHARMACOKINETICS
SO- <PY> 1991
SO- <VO> 20
SO- <IS> N6
SO- <PG> 497-508
LA- ENGLISH
DT- REVIEW
AB- The CNS effects resulting from the combined administration of midazolam and flumazenil were studied in 8 healthy volunteers to develop a model of the pharmacokinetic-pharmacodynamic interaction. Electroencephalograms (EEG) were recorded between Fp1-M1 and Fp2-M2. The EEG parameter total number of waves between 12 and 30Hz (TNW12-30) derived by aperiodic analysis was used to quantify the effect. Following a 15 min baseline EEG recording, infusion of placebo or flumazenil was started. Infusion regimens for flumazenil were designed so that 'steady-state' concentrations of 10 and 20-mu-g/L were obtained. Doses of midazolam 15, 30 and 60mg over 5 min were given 30 min after the start of placebo infusion (session A) or flumazenil infusion to 10-mu-g/L (session B) or 20-mu-g/L (session C), respectively. Venous blood samples were taken until 8h after the start of the flumazenil or placebo infusion. A sigmoid maximum effect (E(max)) model was used to characterise the relationship between the plasma concentration of midazolam which is in equilibrium with the effect compartment concentration (C(e)m) [C(e)m/Kp] and TNW12-30. Within 2 to 5 min of starting the midazolam infusion all subjects fell asleep, with loss of eyelid reflex. They awoke between 25 and 82 min later in all 3 sessions. The mean (+/- SD) plasma drug concentrations of midazolam corresponding to half the maximum increase in TNW12-30 (EC50) were 276 +/- 64, 624 +/- 187 and 1086 +/- 379-mu-g/L in sessions A, B and C, respectively. The half-lives reflecting equilibration between plasma concentration and effect (t1/2k(e)o), estimated by a nonparametric method, were 2.2 +/- 1.2, 3.3 +/- 3.3 and 2.9 +/- 1.2 min for the 3 different sessions. E(max) and N were not affected by flumazenil. In each subject the plot of the average measured steady-state plasma flumazenil concentration versus the EC50 of midazolam showed a linear relationship. The plasma concentration of flumazenil that doubled the EC50 of midazolam (C(f),2) was 6.5 +/- 1.0-mu-g/L. The observed interaction is consistent with the competitive nature of the antagonism of midazolam by flumazenil.

2050298100

10A04

Source: https://www.industrydocuments.ucsf.edu/docs/hlgl0000

THEOPHYLLINE, EEG/EP/ERP ETC, & REVS, BK CHAPS, MONOGRAPHS ETC

FROM MEDLINE {DIALOG: FILE 155}

124

AN- <DIALOG> 08738258
TI- Review: sleep in heart failure.
AU- Yamashiro Y Kryger MH
CS- Department of Respiratory Medicine, University of Manitoba, Winnipeg, Canada.
SO- <JN> Sleep
CP- UNITED STATES
SO- <PY> Sep 1993
SO- <VO> 16 (6) p513-23
SN- 0161-8105
LA- ENGLISH
DT- JOURNAL ARTICLE REVIEW REVIEW, TUTORIAL
RF- 81

2050798101

Source: https://www.industrydocuments.ucsf.edu/docs/hlgl0000

125

AN- <DIALOG> 08617827
TI- Pharmacokinetic-pharmacodynamic modelling in pre-clinical investigations: principles and perspectives.
AU- Danhof M Mandema JW Hoogerkamp A Mathot RA
CS- Leiden/Amsterdam Center for Drug Research, Division of Pharmacology, University of Leiden, The Netherlands.
SO- <JN> Eur J Drug Metab Pharmacokinet
CP- FRANCE
SO- <PY> Jan-Mar 1993
SO- <VO> 18 (1) p41-7
SN- 0398-7639
LA- ENGLISH
DT- JOURNAL ARTICLE REVIEW REVIEW, TUTORIAL
AB- A new approach to preclinical pharmacodynamic investigations is presented which allows, in addition to information on the nature of the pharmacological properties of new chemical entities, also important quantitative pharmacodynamic information to be derived. A single intravenous dose is administered to chronically instrumented rats and the time course of the pharmacological effect is determined in conjunction with plasma or serum concentrations. Datasets obtained in this way are subjected to simultaneous pharmacokinetic/pharmacodynamic modelling to obtain estimates of pharmacodynamic parameters such as EC50, Emax, Hill factor and the rate of biophase equilibration. The new approach was applied in studies with benzodiazepines, baclofen, antiepileptic drugs and adenosine receptor agonists and antagonists. In these studies quantitative EEG parameters, the threshold for convulsion as determined by direct cortical stimulation and various haemodynamic variables were used as a pharmacodynamic endpoint. For drugs exhibiting agonistic or inverse agonistic properties, realistic estimates of the potency and intrinsic efficacy could be obtained by modelling of the direct effects in the various effect models. Estimates of the potency of competitive antagonists (e.g. flumazenil, cyclopentyl-theophylline) could be obtained on the basis of modelling of the pharmacodynamic interaction with a full agonist. For baclofen finally also the rate of biophase equilibration could be estimated. The results of these studies show that by implementation of pharmacokinetic-pharmacodynamic modelling in pre-clinical investigations, useful quantitative information on the pharmacodynamics of new drugs in vivo can be obtained. It is suggested that this information may be of value in the early phases of pre-clinical drug development and that it may facilitate the subsequent clinical development process.
RF- 17

2050798102

10A04

Source: https://www.industrydocuments.ucsf.edu/docs/hlgl0000

126

AN- <DIALOG> 08382638
TI- Advances in syncope: a combined approach utilizing head-up tilt testing and electrophysiologic evaluation.
AU- Hoch DH Rosenfeld LE
CS- Yale University School of Medicine, New Haven, CT 06510.
SO- <JN> Conn Med
CP- UNITED STATES
SO- <PY> Oct 1992
SO- <VO> 56 (10) p515-23
SN- 0010-6178
LA- ENGLISH
DT- JOURNAL ARTICLE REVIEW REVIEW, TUTORIAL
AB- The electrophysiological evaluation of syncope of unknown origin yields a diagnosis in approximately 40% of patients. In the presence of structural heart disease ventricular tachycardia is the most common etiology accounting for 20% of cases. Over the past several years head-up tilt table testing with isoproterenol provocation has highlighted the syndrome of neurocardiogenic syncope. This syndrome accounts for an additional 30-40% of patients with syncope. There is compelling evidence that this syndrome involves the Bezold-Jarisch reflex with excessive stimulation of ventricular mechanoreceptors (C-fibers) located predominantly in the inferoposterior portion of the heart. Tilt table testing is now an established tool both for diagnosis of this syndrome and for guiding therapy with beta blockers, disopyramide, theophylline, or alpha-agonists. Tilt table testing combined with invasive electrophysiological testing significantly increases the diagnostic yield in the evaluation of syncope.
RF- 88

2050298103

Source: https://www.industrydocuments.ucsf.edu/docs/hlgl0000

127

AN- <DIALOG> 07046239
TI- Bioelectrical impedance in clinical practice.
AU- Zarowitz BJ Pilla AM
CS- Department of Pharmacy Services, Henry Ford Hospital, Wayne State University, Detroit, MI 48202.
SO- <JN> DICP
CP- UNITED STATES
SO- <PY> Jul-Aug 1989
SO- <VO> 23 (7-8) p548-55
SN- 1042-9611
LA- ENGLISH
DT- JOURNAL ARTICLE REVIEW REVIEW, TUTORIAL
AB- Bioelectrical impedance (BI) relies on the conduction of a low-voltage alternating current through the body. Lean tissue and fluids containing electrolytes conduct the current and cell membranes serve as capacitors and account for capacitive resistance. Fat and bone are poor conductors. Measurement of the voltage drop of the applied current yields resistance (R) and reactance (Xc). R and Xc are used with height, weight, age, and gender in a number of multiple regression relationships to predict body composition compartments such as fat-free mass, lean body mass, extracellular mass, and body cell mass. The technique has been compared with and validated against traditional measures of body composition analysis. In clinical practice, BI has been used to monitor fluid status in burn and dialysis patients, assess changes of body cell mass with nutritional repletion, and predict pharmacokinetic parameters and dose of theophylline and aminoglycoside antibiotics. BI is a noninvasive, safe, rapid, and reproducible technique with exciting potential in clinical practice.
RF- 45

128

AN- <DIALOG> 04191978
TI- The use of in vitro brain slices for multidisciplinary studies of synaptic function.
AU- Lynch G Schubert P
SO- <JN> Annu Rev Neurosci
CP- UNITED STATES
SO- <PY> 1980
SO- <VO> 3 p1-22
SN- 0147-006X
LA- ENGLISH
DT- JOURNAL ARTICLE REVIEW
RF- 71

2050798104

Source: https://www.industrydocuments.ucsf.edu/docs/hlgl0000

129

AN- <DIALOG> 03341761
TI- The role of cyclic nucleotides in the CNS.
AU- Phillis JW
SO- <JN> Can J Neurol Sci
CP- CANADA
SO- <PY> Aug 1977
SO- <VO> 4 (3) p151-95
LA- ENGLISH
DT- JOURNAL ARTICLE REVIEW
AB- On the basis of the information presented in this review, it is difficult to reach any firm decision regarding the role of cyclic AMP (or cyclic GMP) in synaptic transmission in the brain. While it is clear that cyclic nucleotide levels can be altered by the exposure of neural tissues to various neurotransmitters, it would be premature to claim that these nucleotides are, or are not, essential to the transmission process in the pre-or post-synaptic components of the synapse. In future experiments with cyclic AMP it will be necessary to consider more critically whether the extracellularly applied nucleotide merely provides a source of adenosine and is thus activating an extracellularly located adenosine receptor, or whether it is actually reaching the hypothetical sites at which it might act as a second messenger. The application of cyclic AMP by intrcellular injection techniques should minimize this particular problem, although possibly at the expense of new diffulties. Prio blockade of the adenosine receptor with agents such as theophylline or adenine xylofuranoside may also assist in the categorization of responses to extracellularly applied cyclic AMP as being a result either of activation of the adenosine receptor or of some other mechanism. Utimately, the developement of highly specific inhibitor for adenylate cyclase should provide a firm basis from which to draw conclusions about the role of cyclic AMP in synaptic transmission. Similar considerations apply to the action of cyclic GMP and the role of its synthesizing enzyme, guanylate cyclase. The use of phosphodiesterase inhibitors in studies on cyclic nucleotides must also be approached with caution. The diverse actions of many of these compounds, which include calcium mobilization and block of adenosine uptake, could account for many of the results that have been reported in the literature.
RF- 420

130

AN- <DIALOG> 02253123
TI- On the mechanism of action of cyclic AMP and its role in synaptic transmission.
AU- Greengard P McAfee DA Kebabian JW
SO- <JN> Adv Cyclic Nucleotide Res
CP- UNITED STATES
SO- <PY> 1972
SO- <VO> 1 p337-55
SN- 0084-5930
LA- ENGLISH
DT- JOURNAL ARTICLE REVIEW
RF- 38

2050798105

Source: https://www.industrydocuments.ucsf.edu/docs/hlgl0000

FROM SCISEARCH {ISI's *Science Citation Index*; DIALOG: File 434}

131

GA- KH130
NR- 169
TI- CARDIAC ELECTROPHYSIOLOGY AND PHARMACOLOGY OF ADENOSINE - BASIC AND CLINICAL ASPECTS
AU- PELLEG A BELARDINELLI L
CS- HAHNEMANN UNIV,MAIL STOP 110,BROAD & VINE/PHILADELPHIA//PA/19102
SO- <JN> CARDIOVASCULAR RESEARCH
SO- <PY> 1993
SO- <VO> 27
SO- <IS> N1
SO- <IS> JAN
SO- <PG> 54-61
SN- 0008-6363
LA- ENGLISH
DT- REVIEW

2050798106

Source: https://www.industrydocuments.ucsf.edu/docs/hlgl0000

132

GA- HK868
NR- 91
TI- ANTIINFLAMMATORY STRATEGIES FOR THE TREATMENT OF ASTHMA
AU- BARDIN PG JOHNSTON SL HOLGATE ST
CS- SOUTHAMPTON GEN HOSP,IMMUNOPHARMACOL GRP/SOUTHAMPTON SO9 4XY/HANTS/ENGLAND/
SO- <JN> SOUTH AFRICAN MEDICAL JOURNAL
SO- <PY> 1992
SO- <VO> 81
SO- <IS> N6
SO- <IS> MAR 21
SO- <PG> 303-309
LA- ENGLISH
DT- REVIEW
AB- The inflammatory basis of asthma is now beyond dispute and even mild asthmatics exhibit mast cell degranulation, eosinophil infiltration and increases in mononuclear cells in airway mucosal biopsies. The chronic nature of this endobronchial inflammation may cause damage to ciliated epithelium, which, coupled with laying down of cross-linked collagen within the airway wall, leads to partly irreversible airway obstruction. Corticosteroids, which are potent anti-inflammatory agents, decrease bronchial hyperresponsiveness and the clinical manifestations of asthma. Although inhaled corticosteroids produce fewer side-effects, the use of low-dose (10 mg or less) oral treatment may be recommended for patients unable financially to afford inhaled corticosteroids or who are unable to use them effectively. Other anti-inflammatory drugs, including methotrexate, cyclosporin and the newer leukotriene inhibitors, are not yet in general use and may provide new pharmacological approaches to the treatment of asthma in the near future. In all but the mildest asthma, strategies aimed at preventing and decreasing bronchial inflammation should be the primary aim of treatment. The physician should refrain from prescribing only beta-2-agonists to new asthmatics and patients must be educated to increase understanding of the benefits of preventive rather than symptomatic forms of treatment for this chronic disease.

133

GA- P5593
NR- 332
TI- DRUG-INTERACTIONS THAT MATTER - A CRITICAL REAPPRAISAL
AU- MCINNES GT BRODIE MJ
CS- UNIV GLASGOW,WESTERN INFIRM,GARDINER INST,DEPT MED,CLIN PHARMACOL UNIT/GLASGOW G11 6NT//SCOTLAND/
SO- <JN> DRUGS
SO- <PY> 1988
SO- <VO> 36
SO- <IS> N1
SO- <PG> 83-110
LA- ENGLISH
DT- REVIEW, BIBLIOGRAPHY

2050298107

Source: https://www.industrydocuments.ucsf.edu/docs/hlgl0000

134

GA- RA012
NR- 343
TI- A COLLECTION OF THERAPEUTIC, TOXIC AND FATAL BLOOD DRUG CONCENTRATIONS IN MAN
AU- STEAD AH MOFFAT AC
CS- CENT RES ESTAB,HOME OFF FORENS SCI SERV/READINGRG7 4PN/BERKS/ENGLAND/
SO- <JN> HUMAN TOXICOLOGY
SO- <PY> 1983
SO- <VO> 2
SO- <IS> N3
SO- <PG> 437-464
LA- ENGLISH
DT- REVIEW, BIBLIOGRAPHY

2050798108

Source: https://www.industrydocuments.ucsf.edu/docs/hlgl0000

CARBON MONOXIDE, EEG/EP/ERP ETC & HEALTHY/NORMAL SUBJECTS/VOLS

FROM EMBASE {*Excerpta Medica*; DIALOG: File 73}

135

AN- <DIALOG> 5957699
TI- Apperceptive agnosia due to carbon monoxide poisoning. An interpretation based on critical band masking from disseminated lesions
AU- Campion J. Latto R.
CS- Department of Psychology, University of Liverpool, Liverpool
CS- UNITED KINGDOM
SO- <JN> BEHAV. BRAIN RES.
CP- NETHERLANDS
SO- <PY> 1985
SO- <VO> 15/3 (227-240)
LA- ENGLISH
AB- Apperceptive visual agnosia is normally held to be a specific deficit in 'apperception' - a hypothetical postsensory stage in visual processing. This paper describes the investigation of a patient diagnosed as suffering from a classical apperceptive agnosia resulting from carbon monoxide poisoning. Controlled behavioural testing confirmed the apparent agnosia but revealed that he could be trained to make a number of visual discriminations which had not been apparent from routine clinical examination and that he suffered a number of subtle sensory impairments which likewise had not hitherto been apparent. Evoked potential recording to grating patterns showed a complex pattern of brain responses involving interactions between spatial frequency, orientation and hemisphere recorded from. The data suggested that the agnosia was caused by sensory impairments rather than a deficit in apperception. We proposed that the impairments were caused by loss of certain spatial frequency and orientation information but rejected an interpretation based on the concept of processing channels in favour of one based on object contour masking by a peppery field defect caused by disseminated lesions. This interpretation received some support from fine grain static perimetry, contrast sensitivity function measurement and orientation discrimination in the two hemifields. Qualitatively similar results were obtained in normal subjects whose field was artifically masked. The results have implications for theories of visual agnosia and for theories of vision based on the concept of processing channels.

2050798109

Source: https://www.industrydocuments.ucsf.edu/docs/hlgl0000

136

AN- <DIALOG> 5401117
TI- Moderate carbon monoxide exposure during sleep: neuro- and psychophysiological effects in young and elderly people
AU- Groll Knapp E. Haider M. Jenkner H. et al.
CS- Inst. Environ. Health, Univ. Vienna, A-1095 Vienna
CS- AUSTRIA
SO- <JN> NEUROBEHAV. TOXICOL. TERATOL.
CP- USA
SO- <PY> 1982
SO- <VO> 4/6 (709-716)
LA- ENGLISH
AB- In order to assess age-related effects of carbon monoxide (CO) on brain wave-activity and sleep, auditory evoked potentials (AEP) were measured during sleep in 10 healthy young volunteers (20-25 years) and in a group (N=10) of healthy elderly Ss (55-72 years) under control and exposure conditions in counterbalanced order. Sleep-stages were classified according to the Rechtschaffen and Kales scoring-system. In addition to electrophysiological measures memory-performance and subjective feeling (mood-scale) was assessed before and after sleep. Eight hours of CO-exposure (100 ppm) gave rise to about 8% COHb. In the group of young Ss there was a significant ($p<0.05$) increase of deep sleep (stages 3, 4), and a decrease of stage REM-sleep during CO-exposure, whereas in elderly Ss similar but non-significant sleep-changes occurred. Some CO-related AEP-changes within different sleep-stages, namely shorter latencies in elderly Ss and higher amplitudes in young Ss, approached significance. Within the group of young Ss there was a tendency to more depressed mood after Co-exposure, as well as to Co-related impairment of memory consolidation; no such effects were observed in elderly Ss. The general hypothesis of more pronounced CO-susceptibility of elderly people was, therefore, not supported in this study.

2050798110

Source: https://www.industrydocuments.ucsf.edu/docs/hlgl0000

FROM MEDLINE {DIALOG: FILE 155}

137

AN- <DIALOG> 04669115
TI- Eye movement as an indicator of brain function.
AU- Kojima T Shimazono Y Ichise K Atsumi Y Ando H Ando K
SO- <JN> Folia Psychiatr Neurol Jpn
CP- JAPAN
SO- <PY> 1981
SO- <VO> 35 (4) p425-35
SN- 0015-5721
LA- ENGLISH
DT- JOURNAL ARTICLE
AB- The level of consciousness between the alert and drowsy states was classified into four stages (Alert, Resting I, Resting II, Drowsy) by studying three factors of the EEG patterns on 23 normal subjects. The eye movements recorded by electro-oculograph were divided into two groups, i.e. rapid eye movements (R type, r type) and slow eye movements (S type, s type). The occurrence of each type of eye movements was confirmed to change in close correspondence to the stages of consciousness. The eye movement on 43 cases with a disturbance of consciousness by metabolic disease were recorded longitudinally according to clinical states. The S type movements were predominantly observed in a state of clouding of consciousness, while the R type and R-S type were observed in a delirious state.

2050798111

Source: https://www.industrydocuments.ucsf.edu/docs/hlgl0000

CO & EP/EEG/ERP REVIEWS, BOOK CHAPTERS, MONOGRAPHS, ETC.

FROM EMBASE {*Excerpta Medica*; DIALOG: File 73}

138

AN- <DIALOG> 842046
TI- Neurobiological and behavioural toxicity in animals. (A review of papers presented at the workshop of PC IAOH sub committee on higher nervous functions, Prague, July 1975)
AU- Baettig K.
CS- Inst. Behav. Sci., ETH, Zurich
CS- SWITZERLAND
SO- <JN> ACTIV.NERV.SUP. (PRAHA)
CP- CZECHOSLOVAKIA
SO- <PY> 1976
SO- <VO> 18/4 (270-274)
LA- ENGLISH
AB- Although the application of behavioral methods in environmental toxicology has not yet reached the same level of sophistication as in the field of psychopharmacology the approach seems nevertheless very promising. Different side effects of environmental substances which otherwise might be overlooked, could be detected by a critical application of such techniques. On the other hand much more research will be needed with respect to the standardization of such methods as well as in view of the interpretation of the obtained results and their comparison with the effects on humans.

2050798112

Source: https://www.industrydocuments.ucsf.edu/docs/hlgl0000

FROM MEDLINE {DIALOG: FILE 155}

139

AN- <DIALOG> 08237370
TI- Cross species extrapolation in neurotoxicology: neurophysiological and neurobehavioral aspects.
AU- Winneke G
CS- Department of Psychophysiology, Heinrich-Heine-Universitat Dusseldorf, F.R.G.
SO- <JN> Neurotoxicology
CP- UNITED STATES
SO- <PY> Spring 1992
SO- <VO> 13 (1) p15-25
SN- 0161-813X
LA- ENGLISH
DT- JOURNAL ARTICLE REVIEW REVIEW, TUTORIAL
AB- The theory of phylogenetic continuity of animal species is the basis of any comparative or extrapolative endeavour (Calabrese, 1983). Cross species extrapolation is also a prerequisite for hazard identification in general and developmental neurotoxicology. Two steps must be distinguished: The first step is endpoint-based or qualitative, whereas the second is dose-based or quantitative. Comparison of different species, typically rodents, nonhuman primates and humans, in terms of endpoints is preferentially done within a framework of broad functional categories, such as sensory, motivational, cognitive, motor, and social variables. Within each category specific neurobehavioral as well as electrophysiological measures need to be considered; typically the degree of comparability is higher for electrophysiological than for most behavioral measures. For some frequently used behavioral endpoints in human neurotoxicology, such as psychometric IQ, there is no direct animal counterpart. Once the neural substrate of a particular neurotoxic effect has been identified, as is true for several chemicals such as the pyrethroid insecticides, the organophosphates, most nerve gases or MPTP, or if interspecies comparability in terms of endpoints has proven satisfactory, an effort towards quantitative, dose-based extrapolation is needed. Here species-specific differences in toxicokinetics and metabolism must be taken into consideration in order to arrive at valid translations of dose-response contingencies. If at all possible internal rather than external doses should serve as the frame of reference here. Neurotoxic chemicals of environmental concern for which an adequate data base is available for comparative purposes include alcohol, carbon monoxide, lead, methylmercury and polychlorinated biphenyls (PCB). Principles of cross species extrapolation in neurotoxicology will be illustrated by means of representative neurobehavioral and electrophysiological findings.
RF- 52

2050798113

Source: https://www.industrydocuments.ucsf.edu/docs/hlgl0000

140

AN- <DIALOG> 05739620
TI- Evoked potentials in the assessment of neurotoxicity in humans.
AU- Arezzo JC Simson R Brennan NE
SO- <JN> Neurobehav Toxicol Teratol
CP- UNITED STATES
SO- <PY> Jul-Aug 1985
SO- <VO> 7 (4) p299-304
SN- 0275-1380
LA- ENGLISH
DT- JOURNAL ARTICLE REVIEW
AB- The present paper reviews strengths and weaknesses of evoked potentials (EPs) as an index of toxic insult to the nervous system. EPs are obtained by averaging successive samples of EEG time-locked to the presentation of stimuli. Components of the resulting waveform can be measured for amplitude, latency, and distribution. Normal ranges of these parameters have been characterized for auditory, visual and somatosensory stimuli. Auditory EPs have been studied in humans exposed to lead, trichloroethylene, and carbon monoxide. Changes in timing of short latency components and in amplitude of later cortical components have been reported. Methyl mercury, n-hexane and carbon monoxide cause complex changes in the waveshape of flash and patterned visual EPs. Similarly, specific components of somatosensory EPs are altered following exposure to carbon monoxide, lead and acrylamide. The current lack of standardized recording and analysis techniques has sometimes generated contradictory results, but the evidence thus far supports the ultimate usefulness of EPs as a neurotoxicological screening tool.
RF- 31

141

AN- <DIALOG> 04631940
TI- Physiological aspects of primary hypertension.
AU- Folkow B
SO- <JN> Physiol Rev
CP- UNITED STATES
SO- <PY> Apr 1982
SO- <VO> 62 (2) p347-504
SN- 0031-9333
LA- ENGLISH
DT- JOURNAL ARTICLE REVIEW
RF- 700

2050798114

Source: https://www.industrydocuments.ucsf.edu/docs/hlgl0000

142

AN- <DIALOG> 03628330
TI- Effect of rapid decompression and associated hypoxic phenomena in euthanasia of animals: a review.
AU- Booth NH
SO- <JN> J Am Vet Med Assoc
CP- UNITED STATES
SO- <PY> Aug 1 1978
SO- <VO> 173 (3) p308-14
SN- 0003-1488
LA- ENGLISH
DT- JOURNAL ARTICLE REVIEW
AB- Documentation in the literature indicates that death is as painless following the induction of hypoxia by rapid decompression as by other methods that lead to hypoxia, such as exposure to high altitude, carbon monoxide, and inert gases (nitrogen, xenon, and krypton). Many of the signs and symptoms of hypoxia are the same as those for alcoholic intoxication and inert gas narcosis. Moreover, there is good evidence that analogous relationships or mechanisms may exist for hypoxia, inert gas narcosis, and anesthesia.
RF- 60

143

AN- <DIALOG> 03136284
TI- Applications of neurophysiological methods in occupational medicine. A review.
AU- Seppalainen AM
SO- <JN> Scand J Work Environ Health
CP- FINLAND
SO- <PY> Mar 1975
SO- <VO> 1 (1) p1-14
LA- ENGLISH
DT- JOURNAL ARTICLE REVIEW
RF- 74

144

AN- <DIALOG> 02907391
TI- Effects of moderate CO dose on the central nervous system-- electrophysiological and behaviour data and clinical relevance. pp 217-32.
AU- Haider M Groll-Knapp E Holler H Neuberger M Stidl H
SO- <JN> In: Finkel AJ, Duel WC, ed. Clinical implications of air pollution research. Acton, Mass., Publishing Sciences Group, 1976. WA 754 A514c 1974.
CP- NETHERLANDS
CA- WA 754 A514c 1974
LA- ENGLISH
DT- MONOGRAPH

2050798415

Source: https://www.industrydocuments.ucsf.edu/docs/hlgl0000

145

AN- <DIALOG> 02363633
TI- Neurophysiological effects of hypoxia.
AU- Michael JA
SO- <JN> Monogr Neural Sci
CP- SWITZERLAND
SO- <PY> 1973
SO- <VO> 1 p65-121
SN- 0300-5186
LA- ENGLISH
DT- JOURNAL ARTICLE REVIEW
RF- 216

146

AN- <DIALOG> 01644526
TI- Acute poisoning: some myths and misconceptions.
AU- Matthew H
SO- <JN> Br Med J
CP- ENGLAND
SO- <PY> Mar 6 1971
SO- <VO> 1 (5748) p519-22
SN- 0007-1447
LA- ENGLISH
DT- JOURNAL ARTICLE REVIEW
RF- 36

2050798116

Source: https://www.industrydocuments.ucsf.edu/docs/hlgl0000

ACETALDEHYDE, EEG/EP/ERP ETC & HEALTHY/NORMAL SUBJECTS/VOLS

FROM EMBASE {*Excerpta Medica*; DIALOG: File 73}

147

AN- <DIALOG> 2587778
TI- The calcium carbimide-ethanol interaction: Lack of relation between electroencephalographic response and cerebrospinal fluid acetaldehyde
AU- Hillbom M.E. Lindros K.O. Larsen A.
CS- Res. Lab., State Alcoh. Monopoly, SF-00101 Helsinki 10
CS- FINLAND
SO- <JN> TOXICOL. LETT.
CP- NETHERLANDS
SO- <PY> 1981
SO- <VO> 9/2 (113-119)
LA- ENGLISH
AB- Penetration of acetaldehyde into cerebrospinal fluid (CSF) was studied in healthy human volunteers during calcium carbimide-ethanol interaction. CSF was sampled via lumbar puncture and blood from a cubital vein. CSF and blood acetaldehyde concentrations varied from 1 to 41 and from 22 to 138 mumol/l, respectively. The results indicate that acetaldehyde penetrates the human blood-liquor barrier. Computer analysis of electroencephalograms (EEGs) recorded during the interaction showed reduction in alpha activity with a concomitant increase in delta activity. The changes were similar to those previously observed during 'normal' ethanol intoxication.

2050798117

Source: https://www.industrydocuments.ucsf.edu/docs/hlgl0000

FROM SCISEARCH {ISI's *Science Citation Index*; DIALOG: File 434}

148

GA- NR675
NR- 38
TI- ABSTINENT ALCOHOLICS EXHIBIT AN EXAGGERATED STRESS-RESPONSE TO 2-DEOXY-D-GLUCOSE CHALLENGE
AU- GEORGE DT LINDQUIST T ALIM T FLOOD M ECKARDT MJ LINNOILA M
CS- NIAAA,CLIN STUDIES LAB,BLDG 10,ROOM 3B19,9000 ROCKVILLE PIKE/BETHESDA//MD/20892 NIH,CTR CLIN,DEPT NUTR/BETHESDA//MD/20892 DEPT VET AFFAIRS MED CTR,NATL INST DRUG ABUST,RES UNIT/WASHINGTON//DC/00000
SO- <JN> ALCOHOLISM-CLINICAL AND EXPERIMENTAL RESEARCH
SO- <PY> 1994
SO- <VO> 18
SO- <IS> N3
SO- <IS> JUN
SO- <PG> 685-691
SN- 0145-6008
LA- ENGLISH
DT- ARTICLE
AB- Chronic excessive alcohol consumption can significantly disturb the hypothalamic control of glucose metabolism; however, the mechanism and clinical significance of this disturbance are poorly understood. We used 2-deoxy-D-glucose (2-DG), which produces intracellular glucoprivation, to compare neurochemical, physiological, and behavioral responses to glucoprivic stress between alcoholics abstinent for 3 weeks and healthy volunteers. Twenty-six male alcoholics and 15 male healthy volunteers received intravenous infusions of placebo, 12.5 mg/kg, and 25.0 mg/kg of body weight of 2-DG over 30 min on three separate days, following a random-ordered, double-blind procedure. Minimal effects were observed following administration of the 12.5 mg/kg of body weight dose of 2-DG. Following 25.0 mg/kg, alcoholics showed both exaggerated ACTH and cortisol responses and greater increases in caloric intake when compared with controls. Although anxiety, desire to consume alcohol, plasma progesterone, and sympathetic and adrenal medullary activity all increased following 2-DG, these responses did not differ between alcoholics and controls. The present findings suggest certain specificity for the exaggerated hypothalamic and adrenocortical responses to mild glucoprivic stress in 3-week-abstinent alcoholics.

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Source: https://www.industrydocuments.ucsf.edu/docs/hlgl0000

ACETALDEHYDE, EEG/EP/ERP REVS, BOOK CHAPTERS, MONOGRAPHS, ETC

FROM psycINFO {DIALOG: FILE 11}

149

AN- 00472322
TI- Familial alcoholism: A separate entity?
AU- Goodwin, Donald W.
CS- U Kansas Medical Ctr, Kansas City
SO- <JN> Substance & Alcohol Actions/Misuse
SO- 1983 Vol 4(2-3) 129-136
SN- 01918877
LA- English
DT- JOURNAL ARTICLE REVIEW
AB- Discusses evidence indicating that alcoholism runs strongly in families and that the illness may have a genetic component. This evidence has stimulated 2 lines of research. One type involves comparing alcoholics with and without a family history of alcoholism. The other involves comparing college-age sons of alcoholics with sons of nonalcoholics before members of either group have had an extensive drinking history. Studies show that familial alcoholics differ from nonfamilial alcoholics in having an earlier age of onset and symptoms of greater severity. Familial alcoholism has also been associated with a childhood history of hyperactivity and conduct disorder and an adult history of antisocial behavior. Sons of alcoholics compared to controls have been reported to have higher blood acetaldehyde levels after drinking alcohol as well as more subjective and motor tolerance for alcohol. Sons of alcoholics also generate more EEG alpha activity after alcohol and exhibit lower scores on the Categories test of the Halstead-Reitan Neuropsychological Test Battery. About half of hospitalized alcoholics have a family history of alcoholism. Studies indicate that alcoholics with and without a family history of alcoholism differ on a number of variables. Twin, adoption, and high-risk studies add further evidence that ""familial alcoholism'' is a separate diagnostic entity. (29 ref)
AB- (PsycINFO Database Copyright 1984 American Psychological Assn, all rights reserved)

FROM MEDLINE {DIALOG: FILE 155}

150

AN- <DIALOG> 06612744
TI- The role of genetics in the pathogenesis of alcoholism.
AU- Searles JS
SO- <JN> J Abnorm Psychol
CP- UNITED STATES
SO- <PY> May 1988
SO- <VO> 97 (2) p153-67
SN- 0021-843X
LA- ENGLISH
DT- JOURNAL ARTICLE REVIEW REVIEW, ACADEMIC
RF- 101

2050798119

Source: https://www.industrydocuments.ucsf.edu/docs/hlgl0000

151

AN- <DIALOG> 05600398
TI- Studies of populations at high risk for alcoholism.
AU- Schuckit MA
SO- <JN> Psychiatr Dev
CP- ENGLAND
SO- <PY> Spring 1985
SO- <VO> 3 (1) p31-63
SN- 0262-9283
CN- 5526
LA- ENGLISH
DT- JOURNAL ARTICLE REVIEW
AB- The evidence supporting genetic factors in alcoholism comes from family studies (an alcoholic biological parent is seen in 31 per cent of alcoholics), twin studies (MZ concordance 55 per cent and 28 per cent for DZ twins), and adoption studies (alcoholism 44 per cent higher in adopted out offspring of alcoholics than controls). Once the presence or absence of a biological alcoholic parent is controlled for, rearing experiences and parental loss do not increase the risk for alcoholism. This conclusion justifies the search for genetic factors which might mediate the increased risk, particularly in groups identified as being at high risk for the development of alcoholism. The methodological assets and liabilities of the 'high risk' approach are reviewed, with reference to a detailed discussion of existing longitudinal and cross-sectional studies of high-risk populations. There is little convincing evidence that measurable personality attributes or differences in rate of ethanol breakdown contribute to alcoholism vulnerability, although high risk groups may have a unique EEG pattern in childhood, and in early adulthood decreased intensity of ethanol response, and increased acetaldehyde may be important.
RF- 143

152

AN- <DIALOG> 05549070
TI- [Pathogenesis of alcoholic cardiomyopathy]
TI- <Original> Patogenez alkogol'noi kardiomiopatii.
AU- Frolov VA Dvornikov VE
SO- <JN> Patol Fiziol Eksp Ter
CP- USSR
SO- <PY> Jan-Feb 1985
SO- <VO> (1) p62-7
SN- 0031-2991
LA- RUSSIAN
DT- JOURNAL ARTICLE REVIEW
RF- 102

2050798120

Source: https://www.industrydocuments.ucsf.edu/docs/hlgl0000

FROM SCISEARCH {ISI's *Science Citation Index*; DIALOG: File 434}

153

GA- NB225
NR- 35
TI- P300 DIFFERENCES BETWEEN NONALCOHOLIC YOUNG MEN AT AVERAGE AND ABOVE-AVERAGE RISK FOR ALCOHOLISM - EFFECTS OF DISTRACTION AND TASK MODALITY
AU- BAUER L HESSELBROCK VM OCONNOR S ROBERTS L
CS- UNIV CONNECTICUT,CTR HLTH,DEPT PSYCHIAT/FARMINGTON//CT/06030 UNIV CONNECTICUT,SCH MED,ALCOHOL RES CTR,DEPT PSYCHIAT/FARMINGTON//CT/00000 INDIANA UNIV,SCH MED,ALCOHOL RES CTR,DEPT PSYCHIAT/INDIANAPOLIS//IN/00000
SO- <JN> PROGRESS IN NEURO-PSYCHOPHARMACOLOGY & BIOLOGICAL PSYCHIATRY
SO- <PY> 1994
SO- <VO> 18
SO- <IS> N2
SO- <IS> MAR
SO- <PG> 263-277
SN- 0278-5846
LA- ENGLISH
DT- REVIEW
AB- 1. P300 event-related electroencephalographic potentials were recorded from 79 young adult males, cross-classified with respect to the presence/absence of a family history of alcoholism (FHA) and the presence/absence of a personal history of antisocial personality (ASP) disorder. P300s were elicited using visual and auditory oddball tasks. Each oddball task was repeated with a tracking task added as a distracter. 2. In general, distraction increased the latencies and reduced the amplitudes of P300s elicited by the oddball stimuli. The P300 latency increase occurred only in low risk ASP- and FHA- groups. There was no adaptive increase in P300 latency in the higher risk ASP+ and FHA+ groups. 3. Group differences in P300 were restricted to visual tasks. No interpretable group differences in P300 latency or amplitude were found during the auditory tasks.

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Source: https://www.industrydocuments.ucsf.edu/docs/hlgl0000